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VOLUME LXXXIV.

A

HANDBOOK

OF

PHYSICAL DIAGNOSIS.

COMPRISING THE THROAT, THORAX, AND ABDOMEN.

BY

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London:
THE NEW SYDENHAM SOCIETY.

MDCCCLXXIX.
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LONDON:
PRINTED BY JAS. TRUSCOTT AND SON,
Suffolk Lane, City.

5982

PREFACE TO THE FIRST EDITION.

The design of this handbook is to present a concise description of the various methods pursued in the clinical examination of the thoracic and abdominal organs in health and disease, and an estimate of the diagnostic value of the results so obtained; the examination of the Larynx is treated of in an Appendix.

The general plan of the work, both as regards the order in which the different methods of exploring each organ are discussed and the account of the conditions likely to be met with, is naturally that of a systematic, scientific inquiry.

Our description, however, particularly when dealing with the purely physical methods of examination, will be found to vary considerably in form and fulness, according as groups of similar phenomena, belonging possibly to different diseases, do or do not admit of being explained by *common* causes; in the latter case a *detailed* account of the symptoms has been given. Whenever the nature of the subject permitted, I have endeavoured to preserve a certain uniform order also in the arrangement of details, taking up first the signs obtained by each method of exploration, then their causes, and lastly the physiological and pathological conditions, and the diseases, in which they occur. In this way only was it practicable to include within our relatively narrow limits the large number of pathological phenomena elicited by the various modes of clinical examination. That an account of this kind, indeed, should not be absolutely complete, that it should fail to take cognisance of *every* deviation of the symptoms from the fundamental pathological type, is unavoidable. Though in the course of a long official connection with the Poliklinik of the University of Berlin I have been enabled to lay up a large

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store of observations of this nature, I felt that to introduce them into such a work as the present would be as injudicious as it was unnecessary, as it is only when viewed in relation to the other characteristic symptoms of the affection under consideration that such facts retain their pathological or diagnostic interest; apart from the special case in hand they would not merely burden the text, but the whole work would lose in uniformity and compactness more than it could possibly gain in the value of its contents or in completeness. In the domain of diagnosis, as in that of every other subject of study, the principal object to be kept in view is the arriving at a knowledge of the essential phenomena; these and their physio-pathological causes once known, any departure from the type in particular cases is easily recognised and understood. These considerations have been borne in mind in the preparation of this work. I trust that my task has been, at least to a certain extent, successfully accomplished.

DR. PAUL GUTTMANN.

BERLIN, *Sept.*, 1871.

PREFACE TO THE THIRD EDITION.

THE third edition differs from its predecessors in many respects; not only have important additions and alterations been made in most of the sections, but large parts of several of the principal chapters have been completely re-written. Special care has been devoted to the arrangement of subjects and the order in which their details are discussed, as I have reason to believe, from public criticism, that it is to this feature that the former editions of my handbook owe their favourable reception in Germany and abroad.* May the same good fortune attend the present edition.

DR. PAUL GUTTMANN.

BERLIN, *Oct.*, 1877.

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* Several translations of this work are in circulation on the Continent. The first edition has been translated into Italian by Dr. Clodomiro Bonfigli, Ferrara (Milan, 1872); into Russian by Drs. Nikolaj and Chomjakoff (Kasan, 1872); a second time into Russian by Dr. Wischnewski (Moscow, 1872); and into Spanish by Dr. Luis Góngora (Seville, 1873). The second edition has been translated into French by Dr. F. L. Hahn (Paris, 1877); into Polish by Drs. Kremer and Pareński (Warsaw, 1877); and into Italian by Dr. Bonfigli (Milan, 1877).

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A HANDBOOK

OF

PHYSICAL DIAGNOSIS.

INTRODUCTION.

In the examination of the thoracic and abdominal organs, the methods employed are almost exclusively of a physical character; this is certainly the case with reference to the exploration of the respiratory and circulatory apparatuses, and in part also to that of the abdominal organs. Strictly speaking, the term *physical methods of examination* is applied only to the practice of Auscultation and Percussion; in the wider sense, however, it includes also Inspection and Palpation, as these latter frequently give as direct and valuable information as the two former methods, particularly with regard to certain of the physical properties of the internal organs, such as their consistence, increase in size, and the presence within them of air, fluid, &c. With equal propriety the term may be made to embrace the estimation of the temperature of the body by means of the thermometer, and the measurement of the shape and respiratory movements of the thorax, of the vital capacity of the lungs (Spirometry), of the respiratory pressure (Pneumatometry), and of the arterial pulse (Sphygmography), all these proceedings, the most important of which is Thermometry, being merely delicate aids to Inspection and Palpation, yielding precisely the same kind of information, but with much greater, even with mathematical, exactness. If to these methods of investigation be added the examination of the secretions and excretions of the body, and in some circumstances also that of the blood, we shall have before us all the means which are usually employed in the exploration of the thoracic and abdominal organs, and which we propose to describe in the following pages.

It is obviously unnecessary to make use of all of these methods of examination in every case; some of them, indeed, are of but limited practical application. The mode of procedure depends chiefly on the organ affected; thus, diseases of the respiratory and circulatory apparatuses are recognised principally by percussion and auscultation, those of the abdominal organs by palpation, those of the kidneys by examination of the urine, &c. These means taken singly, however, though of greatest importance in the particular case in hand, do not exhaust all the sources of information open to us; and it is desirable, especially in complicated cases, to have recourse also to the other forementioned methods, in order to secure a full and complete investigation. The diagnosis gains in certainty and precision as the number of available methods of examination, and of the resulting pathological indications, is increased.

It is, further, an essential part of a systematic pathological inquiry that a certain uniform order be observed in the application of the different methods; the natural order is that first the general physical signs, and then the special, be taken up, as, without a knowledge of the former, the latter are apt to be misunderstood, and to lead to erroneous conclusions. The examination should begin, therefore, with the general *inspection* of the body: the practised eye often discovers in this way a multitude of signs, which not merely declare which of the organs is at fault, but frequently reveal the nature and stage of the disease with absolute accuracy. Then only should the special examination be proceeded with. This ought never to be limited to the particular organs or parts concerning which the patient makes special complaint, as his sensations are often excited by purely adventitious circumstances. In the graver class of cases the condition of all the organs must be ascertained: the physician thus frequently lights upon disorders of whose existence the patient, from the absence of subjective symptoms, had no conception, or complications are discovered without the knowledge of which a true understanding of the nature of the disease, or the formation of a sound diagnosis, is impossible. The pathology of the thoracic and abdominal organs presents numerous examples of the intimate relation which the various diseases of these parts bear to each other, and of the importance of keeping this pathological connection before the mind in forming an opinion as to the origin and nature of the morbid process under investigation.

GENERAL EXAMINATION.

THE symptoms of disease of the thoracic and abdominal organs fall naturally into two groups, general and special: in the first are included those which are caused by the reaction of the local disturbance on the system as a whole, and which may be common to the most diverse disorders, in the second those which are characteristic of disease of a particular organ, or even of the precise form of disease by which it is affected.

One of the most prominent symptoms of acute diseases throughout their whole course, and one which appears also at various stages of many chronic affections, is

FEVER.

The chief indication of the presence of fever is *a rise in the temperature of the body*.

In estimating temperature the *Centigrade* (Celsian) thermometer is that in most general use, except in England and America, where *Fahrenheit's* scale has the preference. Should the accuracy of the thermometer not be guaranteed by the name of the maker it must be tested by comparison with a standard instrument. Where circumstances do not permit of this it may be verified with sufficient exactness for practical purposes by simply taking the temperature of one's own body, or that of another healthy person; as we know that this amounts to about 37·3° C. (99·14° F.), a thermometer which gives this result on being tried several days in succession, each day about the same hour, may be pronounced reliable. Even inaccurate instruments may be employed when the exact amount of error is known, allowance being always made for this in reading the scale. The best makers generally give with each thermometer a guarantee of its accuracy, or of the precise degree of difference between it and a standard instrument. The medical thermometer is fitted with but a small scale, as all the possible variations in the temperature of the human body take place within comparatively narrow limits; thus, it is only in very exceptional cases that a point lower than 33° C. (91·4° F.) or higher than 43° C. (109·4° F.) is reached. The degrees are divided into tenths, but differences of even a twentieth are usually quite appreciable. This is precise enough for ordinary use, but for special scientific investigation it is desirable to have a more finely divided scale.

The temperature is most commonly taken in the axilla, occasionally in the rectum or vagina. The bulb of the thermometer should be introduced beneath the border of the pectoralis major, the arm brought close to the side of the body, and the forearm across the breast; the patient then, with his other hand, either keeps the instrument in position or supports the arm which is pressed against the chest. In the cases of children and weakly patients the attendants must look to the fixation of the instrument. The thermometer must remain in position 15—20 minutes before the mercury can be said to have reached its utmost height, as it is only when the arm is brought into contact with the side of the thorax that the axilla becomes a closed cavity; its temperature therefore rises slowly to that of the interior of the body. It thus serves no purpose to warm the instrument before using it; time may be economised, however, by causing the patient to close the axilla shortly before putting in the thermometer, 4 or 5 minutes being then sufficient for making an observation, which is nearly as rapidly as it can be done in the rectum or vagina. This shorter method may be conveniently employed when observations have to be frequently repeated, as, for instance, every 3 or 4 hours. In special cases, when, on account of the surroundings of the patient, the numbers on the scale can not be read off with accuracy, a *self-registering* thermometer becomes indispensable. In this instrument the column of mercury is divided into two parts by a small bubble of air, the upper portion, the index, being one centimeter in length. When the bulb is warmed the mercury expands, carrying the air bubble and index before it, and leaving them behind when it contracts on being removed from the axilla; the position of the index then indicates the maximum temperature reached. Before using the instrument again it must be gently shaken, so as to drive the index towards the lower end.

The temperature obtained in the closed axilla is that of the interior of the body and of the blood; it amounts to 37°—37·3° C., or 0·2°—0·4° C. more in the rectum or vagina. This does not represent the temperature at every period of the day, but only the average for the whole 24 hours. The temperature of healthy persons is subject to a *daily fluctuation*, entirely independent of external circumstances, *rising continuously from morning till evening, and sinking again from evening till morning*. It is at its lowest, 36·5°—36·7° C., at two hours after midnight, remaining about the same point till early morning; it then rises slowly and constantly till 4 or 6 o'clock in the afternoon, when it is at its highest, 37·5°—37·6° C.; this temperature is maintained for a short time, when it again sinks, at first slowly, afterwards more quickly, to the morning minimum immediately after midnight. Thus *the difference between the morning minimum and the evening maximum amounts to about* 1° C. Besides these daily oscillations the temperature of a healthy individual shows other slight variations connected with different conditions of the body. *Exercise* according to its violence and duration, *raises* the temperature to the extent of several tenths, or even of a whole degree; the *taking of food* has a similar effect, whilst the conditions of sleeping and waking appear to have no influence whatever on the heat of the body. We also find slight differences in the average temperature at different ages; it falls 0·1°—0·2° C. from childhood to middle age, rising again later in life. Sex exercises no influence on temperature.

Of the various fluctuations to which the temperature of healthy persons is subject the most important are the daily periodical oscillations, these being also distinctly appreciable in cases of fever, when patients are incapable of such muscular exertion as would be necessary to produce any change of temperature. The daily range of the temperature in febrile affections is frequently much more extensive than under ordinary circumstances, so that it is desirable, in order to be able to estimate it and the varying intensity of the fever at their proper value, to make at least two observations daily; in severe acute diseases, however, when the height of the temperature furnishes indications for the adoption of certain therapeutical measures (such as cold bathing), and when the effect of these has to be carefully noted, the temperature must be taken at intervals of 3 or 4 hours.

Though the methodical use of the thermometer in febrile diseases dates only from 1851 and 1852 (Traube, v. Baerensprung, Wunderlich, &c.), many thermometric investigations had been carried out in the previous decade, and certain important observations, such as the elevation of temperature which accompanies rigors (de Haën), the effect of cold bathing on the temperature in typhus (Currie), had already been made in the previous century.

The temperature may be considered febrile when it rises more than half a degree C. (nine-tenths of a degree F.) above the normal point for the time of day at which it is observed; as, for instance, a morning temperature of 37·5°—38° C., or an evening temperature of over 38° C. taken in the axilla. Such a slight deviation from the standard of health is known as *subfebrile;* when the temperature reaches 38·5° C. it is that of *slight fever,* above 39·5° C. that of *moderately high fever,* over 40° C. that of *high fever.* The highest recorded temperature, which occurred in a case of tetanus, is 44·7° C. (112·4° F.), rising after death to 45·4° C. (113·7° F.). In the most severe acute diseases (pneumonia, typhus, scarlatina, &c.), a higher temperature than 41·5°—42·5° C. is seldom met with, except in cases of sunstroke and relapsing fever.

The march of the temperature of the body in acute diseases presents *three* distinct types.

1. Simultaneously with the commencement of the disease, which is usually announced by a rigor, the temperature begins to rise rapidly and continuously to a certain height, 39°—41° C.;

at this point it remains several days, showing only the ordinary daily fluctuation of 0·5°—1° C. Under favourable circumstances the temperature then falls almost as speedily and continuously as it rose, usually within 12, 24, or at most 36 hours, to the normal point or even slightly lower, in the latter case to return again to the temperature of health when convalescence is established. The best example of this type is fibrinous pneumonia in the adult, in which defervescence begins about the seventh day, seldom earlier. The transition from the temperature of disease to that of health after this manner is termed *Crisis*. These three well-marked stages in the course of the temperature, namely, that of rapid increase (*stadium incrementi*), that in which it is stationary (the *acme*), and that in which it sinks to the normal point (*stadium decrementi*, or *crisis*), are clearly defined in very many acute diseases, though in some the periods are shorter, in some longer, than in pneumonia. Febrile conditions in which the temperature during the acme shows little or no variation for several days receive the name of *continued fever*.

2. In many other acute diseases the temperature rises more slowly, so that several days elapse before it attains its maximum; this rise may be continuous, the normal daily fluctuations, however,—slight increase from morning till evening, slight decrease from evening till morning,—being still traceable, or it may be interrupted. The highest point being reached, it is followed by the period named *acme* or *fastigium*, lasting days or weeks, and characterised by much greater daily variations than are found in health. These *exacerbations* and *remissions* resemble those of the normal range, inasmuch as they occur at the same times—morning and evening; the exacerbation is usually at its height in the afternoon or evening, the remission at its lowest point in the early morning hours. The daily difference amounts to 1°—2·5° C., or rarely 3° C. When the disease has a favourable issue the return to the normal temperature is *gradual*, and takes place either in a continuous descending line (that is, without the evening exacerbations), or in such a way that both the morning and evening temperatures are each day lower; or the morning remission may be well-marked, while the evening exacerbation remains, but becomes smaller on each day and thus slowly approaches the morning temperature. This gradual re-establishment of the normal temperature, which is accomplished in 3—7

days, is designated *Lysis*, as distinguished from rapid defervescence or *Crisis;* and the febrile conditions indicated by a stage of acme, or fastigium, marked by the above-mentioned oscillations, are named *remittent fever*.

3. The third typical course which may be taken by the temperature is found in *intermittent fever*. The paroxysm begins suddenly, usually with a shivering, and the temperature speedily rises to a height otherwise reached only in the most severe acute diseases, 41°—41·5° C., or even higher: in a few hours it sinks again as quickly and continuously to the normal point. On the third day (tertian type), less frequently on the second day (quotidian type), that is after 24 or 48 hours, the same phenomena are repeated at the same time. When the febrile paroxysm is later by a few hours (postponing type) the quotidian becomes tertian; and similarly, when it is earlier by a few hours (anticipating type) the tertian becomes quotidian. The least common type is the quartan, in which the fever returns at the end of 72 hours. In the intervals of exemption from fever (apyrexia), the temperature and general condition of the patient are perfectly normal.

In other cases, of which the type is *relapsing fever*, the febrile attack lasts longer, the temperature mounts to 41° C. or higher, and returns to the normal point in a continuous line. The intervals between the seizures have not the same well-defined quotidian or tertian character as in intermittent fever, but vary in duration from days to weeks. The last-named temperature-types, the intermittent and relapsing, are sometimes closely simulated by the occurrence of sudden exacerbations in the course of various acute diseases, depending on an extension or complication of the local affection; in these cases, however, the temperature in the intermissions is never that of perfect health.

The march of the temperature in *chronic* diseases, when they are accompanied by fever, resembles that of the remittent type, with morning remissions and evening exacerbations, the morning temperature being slightly above the normal, the evening temperature indicating a considerable degree of fever. In another class of cases the type is intermittent, the morning temperature being normal, that of the evening high; more seldom the intervals of apyrexia are of one or more days' duration, during which time both morning and evening temperatures show no deviation from the healthy standard. Both these temperature-types are frequently met with, as in the caseous pneumonia which leads to pulmonary phthisis and in the chronic inflammatory diseases of the abdominal organs.—There remains to be mentioned a somewhat rare form of temperature course, the *typus inversus*, occasionally observed in phthisis and other diseases; in it the remission takes place in the evening, the exacerbation in the morning.

A second sign of fever is *acceleration of the pulse.*

The pulse, in a healthy adult, beats at the rate of 60—80 times per minute, averaging 72; in fever its frequency varies from 80—150. A pulse of 100 may be regarded as indicating slight fever, 100—120 moderately high fever, above 120 high fever; a pulse of 140—150 is rare, even at the crisis of acute diseases, and generally warrants a grave prognosis. Above 150—160 the pulse becomes very small and thready, and is usually the immediate precursor of death in acute organic or zymotic diseases. A pulse of 150—160 occurs, in non-febrile affections, only in certain very rare cases of exophthalmic goître (Basedow's or Graves' disease *).

The acceleration of the pulse is for the most part due to the increased heat of the body. This view is supported by the fact that when animals are exposed to a great heat the heart contracts more rapidly, and when they are cooled again it acts more slowly; and further, at the onset of a fever the rise in temperature often precedes the acceleration of the pulse, and, in the same way, in rapid defervescence the pulse does not become slower till the temperature has first begun to fall. The elevation of temperature, however, though the most important, is not the only cause of the acceleration of the heart's action, as a given alteration of temperature is not always associated with a corresponding change in the pulse; and different individuals, having the same temperature, often show great differences in their pulse-rates. Those forces which in health produce a quick pulse,—such as muscular exertion, mental irritation,—operate much more powerfully in disease, and in that way tend to disturb the relation which undoubtedly exists between temperature and pulse. The same result may also follow the occurrence of certain morbid complications : thus on the accession of an affection of the base of the brain we may have a high temperature with a slow or even subnormal pulse, evidently in consequence of irritation of the origin of the vagus ; and a rapid pulse with a relatively low temperature is frequently observed in cardiac diseases, whether primary or appearing in the course of another affection. Further, in collapse and shortly before death the temperature may fall while the pulse rises. It is thus obvious that the frequency of the pulse cannot be regarded as a trustworthy measure of the intensity of a fever; nevertheless, apart from complications, the influence of a febrile temperature on the pulse is thus far manifest, that when a considerable alteration of temperature takes place the pulse never remains entirely unaffected, but invariably responds to a certain extent. The frequency of the pulse in fever, like the temperature, is subject to periodical daily fluctuations, having a morning minimum and an evening maximum ; and, other conditions being left out of consideration, the extent of this daily difference bears a certain proportion to that of the oscillation of the temperature, so that according as the remissions and exacerbations of the latter are more or less marked the frequency of the pulse is more or less affected. The subsidence of the febrile pulse also, like that of the temperature, may assume the form of crisis or of lysis ; in the *former* case it occurs rapidly, simultaneously with the sinking of the temperature, and may reach the normal point at the very beginning of convalescence, while in the *latter* case it takes place gradually,—sometimes continuously, but more often interruptedly,—with a slight rise each evening.

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* The unusual rapidity of the pulse in this affection is most probably referable to irritation of the sympathetic. Cases of *compression of the vagus* by cervical or intrathoracic tumours are characterised by a very rapid pulse, the cardio-inhibitory function of that nerve being suspended by the pressure. I have, in one instance in which the vagus was involved in a swelling of the lymphatic glands, noted a pulse of 160 accompanied by no elevation of temperature.

Besides elevation of temperature and acceleration of the pulse, changes in the *urine* rank as signs of fever ; these, however, will be discussed further on.

INSPECTION.

MANY diseases of the chest and abdomen, acute as well as chronic, are recognisable at the first glance by a *change in the colour of the skin*. The most common of these changes is

BLANCHING.

A high degree of pallor has always a pathological significance; in a less marked form, however, (as it is often seen in perfectly healthy persons), it can be so regarded only when the symptoms of the disease, subjective and objective, offer a satisfactory explanation of its presence. It attracts attention most readily on those exposed parts which are normally of a red colour, the face, the mucous membrane of the lips, and the conjunctivæ.

It is caused, in every case, either by a *diminution of the volume of blood in circulation*, by *deficiency in the number of red blood-corpuscles*, or by *an unfilled condition of the capillaries*.

The *decrease in the quantity of the blood* may be *direct*, the result of hæmorrhage from the lungs, stomach, bowels, or urogenital apparatus, or of the effusion of blood or blood-tinged exudation into the serous cavities. Or it may be *indirect*, caused by a deficient supply or malassimilation of food,—as in all febrile affections, in convalescence from severe acute disease, and in many chronic diseases of the alimentary canal and the organs connected with it. Further, we have the same condition produced when the system, through loss of albumen, is deprived of much blood-forming material; instances of this are seen in cases in which there is copious effusion into the pleuræ, pericardium, and peritoneum, in general anasarca and in albuminuria.

Blanching from a *deficiency in the number of the red blood-corpuscles* comes under our notice chiefly in chlorosis and other anæmic conditions (anæmia splenica, pernicious anæmia) and in leukæmia. In chlorosis not only is the total quantity of blood in circulation probably diminished, but the red corpuscles may be reduced to one-half their normal relative number, giving rise to such an extreme degree of pallor as is rarely observed in any other affection; in leukæmia the alteration of the proportion of white corpuscles to red is due both to increase of the former and decrease of the latter. In these diseases, also, owing to the fewness of the red corpuscles, the blood is markedly wanting in hæmoglobin, which may be reduced to one-third the normal quantity; this is especially characteristic of some cases of chlorosis in which, not the number of the red corpuscles, but their hæmoglobin, seems to be lessened.

Independently of any change in the blood, however, extreme paleness of the skin may show itself when, from any cause, *the heart does not contract with sufficient force to adequately fill the arteries and capillaries.* Of the many varieties of pallor which come under this category, the following may be mentioned:—that which is connected with emotional influences such as fear or anxiety, and which is immediately produced by contraction of the arterioles from irritation of the vasomotor nerves; that of syncope, which is due to sudden enfeeblement or even momentary cessation of the heart's action; that which appears in fatty degeneration of the heart, and in those cardiac diseases in which, from engorgement of the pulmonary vessels, the left ventricle contains less blood than normally (*e.g.*, mitral disease). It should be remarked that while it is true that blanching may arise from any one of the above-named causes, it most frequently happens that two or more of them co-operate in producing it.

The skin is sometimes simply white, as in chlorosis, when the patient, provided there be no disturbance of nutrition, has otherwise the appearance of health. At other times it is of a dull earthy colour, at once giving the impression of serious organic disease associated with disordered nutrition and emaciation; this is found chiefly in cases of malarial cachexia of old standing, leukæmia, amyloid and carcinomatous degenerations, and many other diseases of the abdominal organs. The absolute colourlessness of the skin is occasionally varied by the presence of slight cyanosis; this occurs most often with those cardiac affections in which there is a tendency to overloading of the pulmonary circulation, and in various diseases of the respiratory apparatus. Caseous pneumonic infiltration, even in its early stages, is marked by pallor of the face, partly from repeated hæmoptyses, but principally from the remittent fever which accompanies it; the whole countenance may be uniformly pale, or certain spots may be of a bright red colour,—the well-known "hectic flush" of consumptives. In these cases, also, the colour comes and goes rapidly under slight physical or mental excitement, the face being suddenly overspread by a fiery blush, which just as suddenly disappears. These symptoms are most pronounced in young persons with a delicate skin and in whom the disease runs a subacute course.

Another common change in the colour of the skin is

CYANOSIS.

It varies much in intensity, from a light bluish tint to a dark bluish-black coloration. In the extremities, and wherever the skin is most delicate and most vascular,—in the lips, the point of the nose, the eyelids, ears, nails, tips of the elbows, and fronts of the knees,—it is earliest and most distinctly seen ; even when the cyanosis is extreme, and the whole surface of the body is discoloured, it is most marked in the parts mentioned. The mucous membranes have the same bluish hue as the skin. Together with the lividity the superficial veins (in the arm, neck, &c.) are seen to be so overloaded with blood as to stand out like bluish knotted cords. These cases must be distinguished from others in which the change of colour is merely *local*, and which will subsequently be discussed. Cyanosis is always an indication that the blood is deficient in oxygen and surcharged with carbonic acid,—that it is either not completely oxidised in the lungs, or that, on account of passing slowly through the capillaries, it gives off too much oxygen to the tissues and absorbs an excess of carbonic acid from them. It arises from one or both of these causes, incomplete oxidation of the blood in the lungs and passive congestion in the capillaries and veins, in various diseases of the respiratory and circulatory organs.

Diseases of the organs of respiration lead to cyanosis either by preventing the access of air to the lungs or by diminishing the respiratory surface, both causes being usually in operation at the same time. The *former* condition is present in those affections in which there is any obstruction in the air-passages, especially the *larynx* or *trachea*, as in spasm of the glottis, croup, and laryngeal diphtheritis, intralaryngeal tumours in the neighbourhood of the rima glottidis, enlargements of the thyroid body, and acute or chronic bronchial catarrh, in which the free entrance of air is hindered by the swelling of the mucous membrane of the smaller bronchi.

Cyanosis results also from *lessening of the breathing surface*, in those diseases in which *the pulmonary vesicles are filled with infiltration* (as in the stage of hepatization in pneumonia), or are *subjected to pressure from without* (as in pleuritic effusion), or *lose their elasticity* and become *inexpansible* (as in emphysema). In these and similar affections the air is not admitted into the alveoli, and when the whole of one lung is implicated the area available for respiration is reduced by one-half. But the intensity of the cyanosis is not directly proportionate to the extent of lung surface from which the air is excluded. The causes of this absence of direct relation are very various, generally peculiar to each case, and therefore not easily generalised; the only constant circumstance is that the *cyanosis is most marked* when the obstruction to the respiration is *suddenly* set up and when the patient is *strong* and *plethoric*. Thus, pneumothorax following gunshot wound or the bursting of a cavity in the lung causes very considerable cyanosis,—a symptom which is not nearly so noticeable in an equally extensive, but gradually-developed, compression of the lung by pleuritic effusion. There is a similar contrast between the symptoms of the *acute* infiltration of pneumonia and those of the *chronic* infiltrations leading eventually to destruction of the air-cells by condensation or atrophy of the lung tissue. Apart from special individual differences, the explanation of the fact that the slowly-advancing diminution of the respiratory surface in chronic disease is associated with but little lividity evidently is that the healthy lung gradually expands, and in that way comes to supply, to a certain extent, the place of the partially disabled lung. That robust, full-blooded persons, suffering from embarrassment of the respiration, should present a higher degree of cyanosis than those who are anæmic is to be expected, as the more plethoric an individual is the more completely are his vessels—and among them those of the lungs—filled, and it is well known that the oxidation of the blood goes on more slowly when the vessels are distended than when they are partially empty. In phthisis, on the other hand, as the emaciation and decrease in the quantity of blood in circulation keep pace with the reduction of the respiratory surface, the cyanosis is always slight. Cyanosis becomes extreme when, as in the emphysema which accompanies chronic bronchitis, both conditions which tend to produce it are present,—obstruction of the air-passages and diminution of functionally active lung surface.

In vesicular emphysema the lividity of the skin is greatly aggravated by the state of *engorgement of the systemic veins.* In consequence of the emphysematous dilatation of the air-cells the capillaries distributed in their walls are partially or totally obliterated, and thus a considerable resistance is offered to the emptying of the right side of the heart. This difficulty the heart is for some time enabled to overcome by hypertrophy and increase of power of the right ventricle; eventually, however, its muscular fibre becomes the seat of fatty degeneration, its action becomes weaker, the right cavities do not discharge the whole of their contents into the lungs and therefore cannot receive the whole of the blood brought to them by the venæ cavæ, so that a permanent engorgement of the venous system is established, manifesting itself in an overloaded condition of the superficial veins (the cervical and brachial veins, &c.), and in cyanotic discoloration of the extremities.

Cyanosis may further be directly caused by *congenital malformations of the heart* or by *acquired valvular disease.* Of the former the most important are the rising of the aorta from the right ventricle, the existence of openings in the interventricular septum, and generally such malformations as permit of direct communication between the two sides of the heart; but persistence of the foramen ovale or ductus arteriosus does not usually occasion very considerable cyanosis. Mitral insufficiency, contraction of the left auriculo-ventricular orifice, tricuspid disease, and fatty degeneration, being generally accompanied by overloading of the right heart, of the pulmonary circulation, and finally also of the systemic veins, tend to induce lividity; this, however, does not occur so long as the impediment to the circulation is counterbalanced by increase of power and hypertrophy of the heart, for although in these cases the vessels of the lungs are over-filled, sufficient oxygenation of the blood is secured by the greater *rapidity of the respiration.* It is only when the right heart is enfeebled by fatty metamorphosis that it remains constantly overcharged with blood, and the complete emptying of the venæ cavæ is prevented; there is thus developed a state of passive stagnation of the current through the capillaries and veins of the body generally, and cyanosis from the consequent free absorption of carbonic acid and the large quantity of oxygen parted with.

In certain diseases of the *abdominal organs cyanosis* may be a very prominent symptom. This is observed when the diaphragm is forced upwards, and the adequate expansion of the lungs in that way hindered, by the presence of fluid or large tumours in the abdominal cavity (ascites, ovarian tumours, &c.). Cyanosis from such causes, when it does occur, is usually very intense. Lastly, marked lividity may result from partial or complete obstruction of the circulation by *compression* or *obliteration* of one of the *large venous trunks:* in this case the cyanosis is *local*, not general, and confined to the region in immediate connection with the vein implicated. The most common examples of this are seen in the cyanotic hue of the forearm and hand on compressing the median vein as a preliminary to venesection, and of the face in severe attacks of coughing. In the latter instance the increase of pressure within the chest takes effect also on the large intrathoracic veins, so as to render turgid the jugular and facial veins. The lividity following occlusion of the veins of the lower limbs by thrombosis is not of a very marked character, as the engorgement of the vessels is relieved by a dropsical effusion into the subcutaneous tissues, and the circulation in the capillaries of the skin almost completely arrested by the pressure. In the rare cases in which the venæ cavæ are entirely obliterated the cyanosis appears in the upper or lower parts of the body, according as it is the superior or inferior cava which is involved.

The livid hue of the skin in *rigors*, and that produced by the influence of *cold* on exposed parts, are due to the contraction of the superficial arterioles and capillaries, and the consequent retardation of the current of blood through them.

ICTERUS.

This *yellow* discoloration is sometimes perceptible only as a slight golden glittering appearance of the most transparent parts, such as the conjunctivæ; at other times it is citron-yellow, orange, or even green or brownish-green (Melan-Icterus). In all severe cases it is observable on the general surface of the body, differences in intensity at various points arising from variations in the delicacy and normal colour of the skin; thus the skin of the breast, and other parts usually covered, becomes more deeply stained than that of the face or the forearms of working people. When the blood is pressed out of the capillaries of such parts, if the jaundice be very marked, the tissues also are seen to have the same yellowish hue ; so also the mucous membranes, the internal organs, tissues, and fluids, all participate in this discoloration. The perspiration, the urine, and sometimes even the sputa, are similarly tinged, while the fæces generally lose their brown colour and acquire a grey or a light *clay-like* appearance. Icterus almost invariably arises from the presence of some mechanical obstacle to the free flow of bile from the ductus choledochus into the duodenum ; the secretion thus accumulates in the bile-ducts and, when these become over-distended, passes through their walls and is absorbed into the blood. This is known as Jaundice from obstruction or absorption. The most frequent cause of this impeded flow of bile is catarrh of the duodenum, the orifice of the gall-duct being closed by the swelling of the mucous membrane (Icterus duodenalis). Closure of the common duct, or of the hepatic duct, or of several of its smaller divisions (by gall-stones, carcinomatous tumours, cirrhosis, or echinococcus cysts), or decrease of the lumen of the various ducts (as by diffuse catarrh), tend to produce more or less icterus. The most severe form, Melan-Icterus, is met with almost exclusively in acute yellow atrophy of the liver.

The only disease of the respiratory organs which is complicated by jaundice and duodenal catarrh, and that in a mild form, is the so-called *bilious pneumonia*. Icterus appears in the later stages of cardiac diseases when, the heart's action being no longer powerful enough to compensate for the embarrassment due to mitral or tricuspid disease or fatty degeneration, there is congestion of the portal circulation, swelling of the liver, and secondary catarrh of the hepatic ducts. Even in these cases it is never very intense, the skin being merely of a dull yellowish colour, modified, as there is usually also more or less passive congestion of the whole venous system, by a slight tinge of cyanosis.

Jaundice sometimes occurs independently of any mechanical obstruction to the escape of bile into the duodenum, in pyæmia, yellow fever, anæmia, after inhalation of chloroform and ether, occasionally after the exhibition of chloral hydrate, and in new-born children.

This form is named *hæmatogenous* icterus,—as distinguished from the forementioned *hepatogenous* variety,—as it is generally believed to be due to partial decomposition of the red blood-corpuscles and subsequent change of the colouring-matter so liberated (hæmoglobin) into a substance chemically related to the colouring-matter of the bile (bilirubin). This theory is based on the experimental facts that bilirubin may be detected in the urine, or that there may be a slight yellowish discoloration of the skin, after the injection of a solution of the salts of the biliary acids into the veins, these salts having a solvent power over the blood-corpuscles, or after injecting various acids which dissolve their hæmoglobin. Transfusion, or even the mere introduction of a quantity of water into the veins, depriving the corpuscles of their hæmoglobin by the operation of the laws of diffusion, leads to the same result. In old extravasations of blood also the presence of biliary colouring matter has been demonstrated. Notwithstanding this physiological evidence, doubt has lately been thrown on the propriety of retaining in our classification a *hæmatogenous* variety of jaundice, inasmuch as the biliary acids, whose supposed absence from the urine in hæmatogenous icterus and presence in that of hepatogenous icterus have been regarded as sufficient grounds for the assumption of two genetically different forms of the affection, have been found also in the urine in pyæmia (Naunyn), and are stated to exist in traces in all urines (Vogel and Dragendorff); and further, many cases hitherto considered as of hæmatogenous origin, hepatogenous causes not being demonstrable, must now come under the latter category as, even though the liver be anatomically intact, jaundice may be produced simply by nerve-influence, whereby the calibre of the gall-ducts is diminished, the flow of bile mechanically obstructed, and the bile itself absorbed into the circulation.—Lowering of the blood-pressure in the hepatic capillaries causes decrease or even suppression of the secretion of bile, which is then absorbed within the liver (Heidenhain and Lichtheim). Of this nature are the icterus of animals which have been starved, that which is associated with free discharge of bile through a biliary fistula, that connected with closure of the portal vein, and probably also that of the new-born (as after birth the stream of blood coming from the umbilical veins to the portal vein ceases). The jaundice in poisoning by phosphorus, which was formerly thought to be hæmatogenous, is obviously hepatogenous, being caused by catarrh of the duodenum and partial closure of the orifice of the ductus choledochus (Virchow).

PIGMENTATION OF THE SKIN.

From the discolorations of the skin just referred to, which have their origin in a change in the colour of the blood, are to be distinguished other varieties closely resembling them, due to *deposit of pigment in the tissue of the cutis*. An instance of this is seen in the *bronzing* which most usually accompanies disease of the supra-renal capsules (known as Addison's disease), but which sometimes presents itself when these organs are perfectly healthy. It occurs in all degrees of intensity, from dull yellowish brown to bluish black, when it might, at the first glance, be easily mistaken for extreme cyanosis: in two cases which came under my own observation the skin was quite as dark as that of a mulatto or negro. The bronzing usually involves large tracts of skin,—when the disease has lasted long generally the whole surface of the body; in most cases it affects specially the exposed parts (the face, backs of the hands), those which in health are most deeply pigmented (genital organs, nipples), and those which are subjected to pressure or friction (the folds of the axillæ, inner surface of the thighs, &c). Its characteristic feature, which defines it sharply from all similar kinds of staining of the skin, developed suddenly or slowly in conjunction with disorders of the generative system, psychical disturbances, &c., is that the conjunctiva bulbi and finger nails are never implicated. Another peculiarity noticed specially in cases of long standing is the occurrence of *scattered spots of pigmentation* both in the already discoloured skin and on the *mucous membrane of the lips and mouth*. Pigmentation of the internal organs in Addison's disease has not yet been observed. The cause of the bronzing of the surface, and the explanation of its connection with disease of the supra-renal capsules, are still unknown.

Argyria, staining of the skin from the long-continued internal use of nitrate of silver, more rarely from painting the throat with a solution of the same salt (Silvestri, Duguet, Krishaber), is but seldom met with, and consists of a deposit of black granular particles of metallic silver, or of silver compounds, in the cutis.* It is exceedingly like the greyish blue cyanotic hue which is so common in congenital malformations of the heart, but is distinguished from it, as is also the forementioned bronzing of the skin, by the fact that it does not disappear on pressure. It sometimes extends over the whole surface (though it may not be of the same deep shade at every point), is at other times confined to certain regions, especially to the exposed parts, and does not pass away on ceasing the administration of the nitrate of silver. It has no general constitutional effect. The deposit of silver is found not only in the skin, but also in the mucous and serous membranes, and in the internal organs (Riemer, &c.).

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* In one case observed by the author, that of a patient suffering from grey degeneration of the posterior columns of the spinal cord, about 24 grammes of the nitrate of silver had been taken in the course of three years; it was only in the third year that the first indications of argyrosis were noticed.

Pigmentation of the skin in small circumscribed spots comes very frequently under notice. Many of these spots are of artificial origin, appearing after the application of vesicants, sinapisms, irritating ointments and liniments, while others are the sequelæ of exanthematous eruptions, ulcers, &c. Though they may have no direct relation to the disease for which a patient may be under examination, they nevertheless afford much valuable information regarding his pathological history, and supply data which enable us to judge better of the accuracy of his statements.

Pityriasis versicolor is a discoloration of the skin confined to certain regions, and caused by the growth of a parasite (microsporon furfur) in the horny layer of the epidermis. It is found in patches of variable size, slightly elevated above the surrounding surface, irregular in shape, and dull yellow or yellowish brown in colour; its most common seat is on the breast, the back, or upper extremities, more rarely on the abdomen or lower extremities. These patches occasionally peel off spontaneously, or may be easily detached in the form of branny scales, when the skin below is discovered to be almost unchanged. The scales, on microscopic examination, are seen to consist of masses of parasitic filaments, between which numerous roundish spores are arranged in clusters. Pityriasis versicolor is often associated with pulmonary phthisis and other chronic diseases leading to anæmia, but it frequently appears also in those who are in good health.

Similar staining of the skin, though not of vegetable parasitic nature, occurs during pregnancy and in the course of diseases of the female generative organs, such as tumours of the ovaries, uterus, &c. In phthisis pulmonalis and many chronic diseases of the abdominal organs the face often assumes a dull yellowish hue, quite different from that of pityriasis versicolor.

Constitution.—General Nutrition.

The *variety of constitution* and the *condition of the nutrition* of the patient can also, to a certain extent, be ascertained by inspection. A knowledge of the *constitution* with which we have to deal furnishes us with many indications of great prognostic and diagnostic value. Thus, those who are feebly constituted may always be said to be in more danger from an attack of acute disease than those who are robust. This is especially true of acute diseases of the respiratory organs; in the vigorous they generally terminate in complete resolution, but tend to recur or to pass into other forms of disease (as caseous degeneration or the various processes resulting in phthisis pulmonalis) in those of a cachectic disposition. In the same way the bronchial catarrh which sometimes supervenes on measles or whooping-cough in weakly children often ultimately spreads to the alveoli. In adults of unsound constitution bronchitis shows a great tendency to return at intervals, to become chronic, and eventually to develope into vesicular emphysema of the lungs, pneumonia runs a longer course, is imperfectly resolved, or ends in caseous metamorphosis, and pleuritic exudations are only partially absorbed, or become the starting point of new morbid processes. It is often of great importance diagnostically to know the constitution of our patient. It enables us, for instance, to distinguish between the onset of caseous pneumonia and that of a simple bronchial catarrh (two conditions which present much the same physical signs), as those who are of untainted constitution are not at all predisposed to destructive changes in the lungs. In this way also, even before making a physical examination of the chest, we can frequently reassure patients who, on account of having a chronic cough, are apprehensive of falling into consumption, as it is certainly but seldom that healthy non-cachectic persons are attacked by this disease.

Well-nourished individuals have firm muscles, an elastic skin, and an ample deposit of fat in the subcutaneous tissue. This development of fat is usually greatest in those who indulge unduly in the pleasures of the table, but is sometimes present to a very considerable degree even in those who are comparatively abstemious; its occurrence is also favoured by a sedentary habit of life, and to a certain extent limited by active exercise. Women often become stouter after the cessation of the menses; and amongst the lower classes the most common cause of obesity is intemperance in the use of alcoholic liquors.

Emaciation usually begins with the disappearance of the subcutaneous adipose tissue; the skin is thus thrown into folds and wrinkles and becomes less elastic, the epidermis is occasionally cast off in the form of branny scales (*pityriasis tabescentium* in cases of marasmus in children), and the muscles lose in volume and power, so that those who are the subjects of much wasting are for the most part confined to bed. Extreme emaciation is always easily recognisable, though when slight it may be apparent only to the patient himself or his friends. The surest test of this condition, however, is comparative weighing at intervals; in the chronic affections accompanying consumption this method gives the most reliable prognostic evidence with respect to improvement, aggravation, or arrest of the disease.

There is always more or less wasting as soon as the weight of the excreta exceeds that of the food, &c., consumed. Thus it is seen in all diseases which are attended for any length of time by fever (the supply of nutriment being almost stopped and the consumption of albumen increased), in persistent stricture of the œsophagus, chronic catarrh of old standing, dilatation and cancer of the stomach, chronic catarrh and ulceration of the intestines, carcinoma of the liver and other organs, diabetes mellitus, &c. Diseases of the circulation, being almost apyrexial, are unattended by emaciation. In acute miliary tubercle of the lungs there is rapid wasting, chiefly owing to the very high fever which accompanies it; it is most probable also that the wasting which is such a constant and prominent symptom of that form of pneumonia which leads to phthisis pulmonalis is less due to the nocturnal sweating than to the febrile disturbance, as, notwithstanding his almost insatiable appetite, the phthisical patient rapidly loses flesh, but whenever the fever abates, and the progress of the disease is thus temporarily arrested, he at once gains in weight.

Emaciation, apart from its prognostic signification, is of some importance from a diagnostic point of view. Thus, of all the chronic diseases of the lungs, caseous pneumonia is the only one in which it is observed; the other chronic lung affections, however, being almost free of fever, may run their course without being marked by any trace of wasting, though they may give rise to absorption of the parenchyma and to the formation of cavities, and offer therefore the same physical signs as are found in caseous pneumonia; such patients indeed, if only their digestive organs be in good condition, may present every indication of being perfectly well nourished.

The consideration of the changes in the skin and in the general nutrition should always be followed by an

EXAMINATION OF THE SUBCUTANEOUS CELLULAR TISSUE.—DROPSY.

One of the commonest morbid alterations observed in diseases of the chest and abdomen, is *accumulation of fluid* in the subcutaneous areolar tissue (*dropsy, œdema*).

The part so affected becomes swollen, this being always the more marked the more lax the structures invaded,—as in the genital organs. The skin loses its natural colour, and becomes pale, tense, and shining. Pressure with the finger gives the sensation of kneading a doughy mass, and leaves behind a more or less deep depression of the surface. This pitting is produced by the fluid being driven out of certain of the meshes of the subcutaneous tissue into those near it and communicating with it; on the cessation of the pressure the fluid slowly returns and the pit disappears. The obliteration of this pressure mark takes place quickly when the anasarca is slight and recent, but more slowly when it is extensive and of long standing, as in the latter case the skin has almost entirely lost its elasticity from the tension and maceration by the effused fluid. Dropsy is invariably caused by the transudation of the serum of the blood through the veins. There is a certain amount of transudation constantly going on even under normal circumstances, but the fluid is at once absorbed by the lymphatic vessels; it is only when the quantity poured out becomes so great that these vessels cannot carry it off that it accumulates in the cellular tissue.

The causes of the increased transudation of fluid are two,—*undue fulness of the veins and consequent increased pressure on their walls*, or *an abnormally watery condition of the blood* which gives rise to changes in the walls of the vessels and renders them more easily permeable (Cohnheim and Lichtheim). Dropsy arising from the first cause is designated *Passive dropsy*, as it always originates in obstruction of the current of venous blood; that from the latter cause is known as *Hydræmic dropsy*.

Passive dropsy, when not strictly local, makes its appearance first in the dependent parts of the body, and on both sides,—in the ankles and on the dorsum of the feet. At first, also, the swelling disappears during the night when the patient is in the horizontal position, the fluid being absorbed by the lymphatics; it returns in the morning, however, as soon as he leaves his bed. In time it tends to become stationary, does not diminish during the night, and mounts by degrees to the legs, thighs, genital organs and the coverings of the chest and abdomen. To this is frequently added effusion of fluid into the peritoneum, pleuræ, and pericardium. It is after this manner that passive anasarca occurs in cardiac diseases in the stage of compensatory disturbance, in which the venous circulation is embarrassed and engorged on account of the constant overloading of the right heart,—as in mitral insufficiency, stenosis of the left auriculo-ventricular orifice, in cases of fatty heart, in tricuspid disease and (seldomer) in the later stages of disease of the aortic valves. This variety of dropsy not unfrequently appears in advanced vesicular emphysema of the lungs, and here also is due to the difficulty experienced by the systemic veins in discharging their contents into the over-filled right side of the heart; it is usually confined, however, to the feet, ankles, and lower part of the legs. The most common abdominal causes of venous congestion are diseases of the liver (cirrhosis and cancer) and of the peritoneum (tubercle and cancer); as it is the portal circulation which is most immediately affected ascites is the first dropsical symptom, this being followed by œdema of the lower extremities only when the venous current in the inferior cava is impeded. Ascites alone, or followed after a considerable interval by œdema of the lower limbs, points invariably to disease of the abdominal organs, in men usually to cirrhosis of the liver.

In dropsy from the second cause, *hydræmia*, the watery condition of the blood induces morbid alterations in the structure of the vessels themselves of such a nature that the serum of the blood passes through them more easily than in health. Hydræmia depends either on an *impoverishment of the blood in respect of albumen and fibrin*, or on *retention of water in the circulation from arrest of the cutaneous transpiration or diminution of the secretion of urine.* It is chiefly in connection with acute or chronic diseases of the kidneys that the blood is found to be poor in albumen; in those affections also the increased blood-pressure within the renal vessels occasions albuminuria. The dropsy of recent renal disease is [illegible] affections by [illegible] particularly [illegible] lower [illegible] this [illegible] stages of [illegible] marked in [illegible] like the [illegible]

[illegible] insufficient [illegible] as the [illegible]

[illegible] the [illegible]

[illegible]

are most often formed in the saphenous and femoral veins, more frequently on one side only than on both, and if the dropsical effusion be not too abundant the clot may be felt through the skin as a hard prominent cord. The œdema gradually disappears on the complete re-establishment of the circulation. Occasionally the cause of localised œdema of the lower limbs is to be sought for at a point higher than the femoral veins,—in the iliac veins or in the inferior vena cava; here again it may be owing to thrombosis, or to compression by the gravid uterus, by tumours, &c.

In many cases, however, localised anasarca cannot be very easily accounted for. I have under my care a robust, florid, otherwise healthy woman, 40 years of age, who for 10 years has suffered from œdema of the legs, reaching as high as the knees, and which during that time has varied but little, if at all; in another case, that of a previously healthy man, an enormous dropsy of the legs and thighs appeared, subsided after a few weeks, and has not since returned. In both cases, notwithstanding repeated and careful examination, no local or general disturbance that the symptom could be traced to was detected; the urine was free of albumen.

Dropsy of one or both *upper* limbs occurs but seldom, and then it is usually produced by compression of the axillary vein (as by enlarged lymphatic glands) or of the subclavian vein. Still more rare is anasarca strictly confined to the upper half of the body; it depends, as a rule, on thrombosis or compression of the vena cava superior, as by intrathoracic tumours or exudations, or aneurism of the aorta.

In the case of a woman 30 years of age, formerly in the enjoyment of good health, I observed dropsy of the upper half of the body, extreme cyanosis, and enormous dilatation of the veins connected with the vena cava superior, all these symptoms being fully developed within three weeks; the lower part of the body remained perfectly normal. Several of the superficial veins of the neck and back appeared to contain only coagulated blood. As no post-mortem examination could be obtained it can only be conjectured that the origin of all these phenomena must have been constriction of the superior vena cava by pressure from without or by thrombosis, as physical examination of the internal organs did not reveal the presence of the slightest morbid change.

A second and rarer abnormity in the subcutaneous tissue is the accumulation of air within its meshes,

Subcutaneous Emphysema.

Like dropsy, it gives rise to a certain amount of swelling, but as this is never so considerable as in that affection, the appearance of the skin is not altered. Emphysematous parts, like those which are anasarcous, pit on pressure with the finger, but not so deeply, nor does the mark last so long, as the elasticity of the skin is almost unimpaired, emphysema being a condition which is generally very rapidly developed. Its most characteristic feature is *the feeling of crackling* or *crepitation* communicated to the hand on pressing the inflated parts, which exactly resembles that experienced on pinching a portion of healthy lung between the fingers. Emphysema is very variable in its distribution, occupying sometimes a smaller, at other times a larger, area, and occasionally extending over nearly the whole of the body. After gaining entrance the air may diffuse itself indefinitely in the subcutaneous cellular tissue, as each mesh communicates with those around it on all sides; this may be demonstrated experimentally on animals, in which it is well-known that the inflation of the entire subcutaneous areolar tissue may be effected from any part of the body.

Subcutaneous emphysema is usually caused by internal or external injury of organs which contain air. Rupture of the œsophagus in the neck by perforating ulcers, from necrosis or the swallowing of foreign bodies, may produce it by allowing the air to enter the cellular tissue at that part, whence it may spread to the breast or further; but when the trachea or one of the larger bronchi is at the same time involved in the ulcerative process and so made to communicate with the œsophagus (broncho-œsophageal fistula), emphysema is wanting. When, in cases of perforation of the stomach or bowel, the affected part becomes attached to the abdominal wall, the gas passes from these organs into the cellular tissue if the opening be large enough; where this attachment does not take place the gas is forced into the peritoneal cavity. Perforation of the larynx and trachea from ulcer gives rise to emphysema in the region of the throat, wounds of the costal pleura and surface of the lungs (by stabbing, gunshot, or fractured ribs, and occasionally by the bursting of an abscess of the lung) to subcutaneous emphysema of the chest. In the latter case the air escapes from the ruptured alveoli or smaller bronchi at each inspiration and passes directly through the torn costal pleura to the subcostal areolar tissue when there are inflammatory adhesions between the injured part of the lung and the chest wall; when no such adhesions exist pneumothorax is first developed, the air being subsequently driven from the pleural sac through the wound in the costal pleura, and so into the cellular tissue.

Emphysema may also be set up by rupture of the air-cells, not from external injury but from over-distension; here the air is forced into the interlobular septa, through the mediastinum, and into the subcutaneous tissue of the neck. It appears first in the fossa jugularis, then in the areolar tissue of the side of the neck, and finally on the surface of the chest. Weakness of the texture of the lung, forcible expansion of the air-cells in severe dyspnœa, violent attacks of coughing, evidently predispose to such tearing of the alveoli, as most of the cases of this variety of emphysema are observed in connection with croup, diphtheritis of the larynx, hooping-cough and bronchitis in children, and advanced pulmonary emphysema in the aged.

Sclerosis of the subcutaneous areolar tissue is a morbid change which is very rarely met with. The extent of surface involved is very variable; thus, of four cases which I have seen, occurring in adults, in one the skin of both lower limbs and of the abdomen was affected, in two that of the arms and face, while in the fourth it appeared in small isolated spots on the forearms and hands. The indurated skin was in all the cases slightly cyanotic, obviously from compression of the smaller cutaneous veins. The pathogenesis of scleroderma is still very obscure. In three cases examined by the author the internal organs were perfectly normal; in a fourth the patient suffered also from Addison's disease, as in another case observed by Rossbach. In a case of almost general scleroderma Heller found small fibroid tumours and largely dilated lymphatic vessels in the areolar tissue under the skin, and obliteration of the thoracic duct; this gives a certain amount of support to the theory that scleroderma depends on some disease of the lymphatic vessels by which the flow of lymph is impeded.

more evident in the greater enlargement of the chest and the displacement of neighbouring parts, the diaphragm and the organs immediately beneath it (the liver on the right, the spleen on the left) being forced downwards, the mediastinum towards the opposite side, and the heart, when the effusion is into the left pleura, towards the right side. Thus the long and transverse diameters are greatly increased, even more than is apparent from mere inspection. Should the fluid be absorbed within a short time the chest usually returns to its normal dimensions, but only when the lung recovers its natural expansibility and elasticity.

Accumulation of air in the pleural sac (pneumothorax) has the same effect on the external conformation of the chest as pleuritic effusion, but as it is, as a rule, suddenly developed, either from external injury to the pleura (by gunshot, stabbing, or fractured ribs) or from the bursting of a superficial pulmonary cavity communicating with one of the larger bronchi,* the lung collapses more quickly and completely, and the affected side soon assumes the form which it has after deep full inspiration. *Actual enlargement* occurs only when the air irritates the pleura and exudation takes place, and the pneumothorax becomes a pyo-pneumothorax. If the case progress favourably, and the offending matters pass off by absorption, which does not often happen, the chest regains nearly its normal shape; most usually, however, the original causes of the pneumothorax bring about a fatal termination.

Extensive pneumonic infiltration of the whole of one lung is occasionally accompanied by considerable enlargement of the corresponding side of the chest. Vesicular emphysema of *one* lung can scarcely ever occur to such a marked degree as to alter the form of the thorax on that side; the author, at least, has never seen such a case.

Tumours within the chest (mediastinal tumours, &c.) may also produce very perceptible prominence of the affected side, and of the sternum. Bulging of the lower part of the thorax on the right side is generally owing to tumour of the liver (hydatid, &c.), on the left to swelling of the spleen, on both sides to hypertrophy of both organs or to distension of the abdomen by ascites, gas, or ovarian tumour.

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* In some rare cases Pneumothorax has occurred after an empyematous effusion had made its way into one of the bronchi, after the bursting of emphysematous air-cells (Rheder), and after rupture of the œsophagus or stomach into the pleural cavity (Heubner, &c.).

Bilateral enlargement of the thorax is often observed in marked and advanced emphysema of the lungs; in typical cases we have the *barrel-shaped chest*, all whose diameters,—its length, breadth, and depth—are increased, the parietes being more prominent both in front and behind, the ribs and sternum more convex, the intercostal spaces wider but not puffed out to the general level of the rest of the surface. This change takes place in the upper and middle parts of the chest, the lower portion usually remaining flat and of the normal form. In other cases this distinctive barrel-shape is wanting, the chest appearing to be uniformly enlarged in every part, even the lower portion being abnormally prominent; or the convexity may be limited to the front or, more frequently, to the back of the chest; and, lastly, there are also certain cases of emphysema in which the thorax undergoes no alteration whatever in size.

These differences in the configuration of the thorax in emphysema depend on the degree of intensity of the affection, the extent of lung implicated, and the exact locality of the lesion (upper or lower, anterior or posterior, parts of the lung); the duration of the disease and the more or less yielding character of the thoracic walls also play an important part in determining these changes, so that they are more common, *cæteris paribus*, among the young than in the aged.

There is only a trifling difference in the circumference of the emphysematous chest in inspiration and in expiration, on account of the very slight amount of expansion and contraction which takes place in the lung; even after full expiration the thorax is abnormally distended, for which reason the typical emphysematous shape has been named the *permanent inspiratory position of the thorax.*

The enlargement of the chest in vesicular emphysema arises from the fact that the lung becomes inelastic and does not subside naturally in expiration, the parietes of the thorax therefore, being no longer called upon to execute the usual movements of respiration, assume permanently the form in question.

The *second group of pathological alterations* in the shape of the thorax include *contraction, diminution in size*, of the whole or part of one side. It occurs most often in connection with the absorption of pleuritic exudation of old standing or when the fluid discharges itself externally. Thus if the lung be subjected to great pressure for months by a large pleuritic effusion its elasticity is more or less completely destroyed and it does not expand on being relieved from this pressure by the disappearance of the fluid,—it is completely void of air and takes up much less room than when fully inflated; the chest-wall, therefore, sinks gradually as absorption goes on. The same explanation applies also to those cases of empyema which burst outwardly. In extreme cases of this nature all the dimensions of the affected side are reduced,—most strikingly antero-posteriorly; there is no trace of bulging at any point, the thorax is flat and sometimes shows on its anterior surface a considerable depression; its long diameter is shortened, the diaphragm being dragged upwards, the ribs made to approach more closely to each other, and the shoulder-blades drawn downwards; its transverse diameter is less, the ribs being forced together, bringing the nipple nearer to the sternum and the shoulder-blade to the vertebral column than on the sound side, while the anterior mediastinum is displaced towards the affected side. Further, there occurs in these cases a distortion of the spinal column, the convexity of which is towards the sound side, the dorsal muscles on the affected side, like the intercostals, becoming paralysed from the persistence of the exudation, so that those of the sound side have no counteracting force opposed to them; in this way also the shrunken side of the chest has its transverse diameter diminished.—The upward displacement of the diaphragm and the dragging of the anterior mediastinum towards the diseased side have an important influence in altering the position of such organs as stand in immediate relation to those parts. Thus, in contraction of the right side the liver rises and the heart encroaches on the right pleuritic cavity; in shrinking of the left side the heart is found situated further towards the left than in health, and above the level of the nipple. The heart's impulse *may*, nevertheless, in cases of sinking of the left side, be felt at a point more or less to the *right* of its normal position, when it has previously been dislocated towards that side by effusion into the left pleura and has there formed adhesions. The most marked contraction of one side of the chest, after the absorption of long-standing exudation or the escape externally of empyematous fluid, takes place in children, because in them we have the difference in growth of the two sides to add to the other differences. The diseased side has its development arrested while the sound side assumes vicarious functions and increases in size accordingly.

I have observed one case, that of a lad 18 years of age, who in childhood had suffered from empyema of the left side which had burst through the skin and discharged outwardly, in which the *left* side was at most but a third of the size of the right, while the heart pulsated below the *right* nipple; it is thus evident that in the early stages of the complaint the heart had been driven over to that position, and had there contracted adhesions.

The shrinking after the absorption of old pleuritic effusion is not always of such a striking character; sometimes certain parts of the lung become again expansible, and then it is only over those portions which remain collapsed that the chest-wall sinks.

The same form of atelectasis as that produced by pleuritic effusion, and the shrinking of the thorax associated with it, might, *a priori*, be expected to occur after the absorption of the gas of pneumothorax of old standing; as the latter disease usually ends in pyothorax, or pyo-pneumothorax, its secondary effects generally coincide with those of pleuritic exudation.

The *third group* of pathological changes in the shape of the chest consists of circumscribed *depressions* of the surface, which are to be distinguished from the foregoing forms of contraction chiefly by their being less considerable in degree and less extensive. They follow shrinking of the lung, from whatever cause it may arise, and owe their existence to the fact that collapsed portions of lung occupy a much smaller space than those which contain air; and as the spare room cannot be filled up by another organ the corresponding parts of the chest-wall yield to atmospheric pressure and form depressions. By far the most common cause of these concavities is caseous condensation of the lungs, and as this takes place most often at the anterior and upper parts, in the supra- or infra-clavicular regions, sometimes on one side, at other times on both, it is in these situations that they may be most confidently looked for. On the lower part of the posterior surface of the chest these depressions are seldom seen, even when there is considerable shrinking of the subjacent lung-tissue, as the powerful dorsal muscles prevent their appearance.

It not unfrequently happens that in chronic interstitial pneumonia and contraction of the pulmonary substance in *children* there are depressions on the surface of the chest of greater or less extent, sometimes even as marked as those connected with pleuritic exudation. In a case attended by me, that of a boy twelve years of age, there was such extreme shrinking of the right lung that the chest showed a very marked depression on that side, reaching as high as the fourth rib; the diaphragm and liver were carried upwards to about same level, while the heart was displaced towards the right side, and pulsated in the third intercostal space close to the sternum.

The abnormities in the conformation of the thoracic parietes which have so far been under discussion are caused by diseases of the respiratory organs. There is another variety, however, which depends on imperfect development, and which itself not unfrequently gives rise to caseous degeneration and tuberculosis of the lungs. It is characterised by a long, narrow, and shallow chest, by sloping of the supra- and infra-clavicular regions, wide intercostal spaces (on account of the diminished power of the intercostal muscles), wing-like projection of the shoulder-blades (from the feeble action of the serrati), undue prominence of the acromial ends of the clavicles, and diminution of the antero-posterior diameter. The manubrium sterni takes part in the flattening, it sinks and so forms an angle (the angle of Louis) at the point where it joins the body of the bone. This is known as the *paralytic* form of thorax, the phthisical habit. Such individuals have an elongated neck, a delicate skin, long extremities, and clubbed fingers; they may, nevertheless, in spite of all these drawbacks, enjoy perfect health, but when attacked by disease of the respiratory apparatus can never feel assured of making so complete a recovery as those who are of a more powerful build.—There is another group of deformities due to diseases of the bones,—to rickets and diseases of the vertebral column. A description of these, however, does not lie within the scope of this work.

MENSURATION OF THE THORAX.

Very slight variations in the form and dimensions of the thorax are easily recognisable even by inspection alone, especially when only one side is concerned. In cases in which such a difference has to be determined once for all a special measurement is scarcely necessary, more particularly as the slighter degrees of difference, which may escape notice on account of the trifling inaccuracies often unavoidable in working with instruments, are distinctly appreciable to the practised eye. When, however, the form of the chest undergoes certain changes in the course of the disease,—when, for instance, one side is first enlarged by pleuritic effusion and afterwards contracts, it may be desirable to ascertain definitely the extent of these variations, in order to obtain a numerical statement of them with which to compare the state of matters at a later period. With this end in view we take the measurements of the circumference and diameters of the chest and the extent of the movements of respiration,—the circumference by means of a *tape* divided into centimeters, the diameters by means of *calipers*.

The *circumference* of the thorax at the level of the nipple in front and of the lower angles of the scapulæ behind, when the arms are raised and outstretched, amounts to about half the length of the body; in well-built men the average is 82 ctmr. (32·28 inches) at the end of an ordinary expiration, and 89 ctmr. (35·04 inches) after a deep inspiration; at the ensiform cartilage the circumference is about 6 ctmr. (2·36 inches) less. In old age it diminishes considerably, especially at the upper part, so that the lower circumference becomes the greater. Perfect symmetry of the two sides of the chest is rare, the right being usually 1—2 ctmr. (0·39—0·78 inch) larger. In measuring only one side of the thorax it is necessary to avoid those sources of error which are apt to arise from differences of attitude on the two sides.

The above numbers are those of Fröhlich, and represent the average circumference in 725 men of 20 years of age, well developed, and destined for the military service. Krug's measurements, made on 3,331 men of 30—34 years of age, agree generally with those of Fröhlich, and show that the girth of the chest varied from 80·9—83·3 cmtr. (31·85—32·79 inches) during expiration, the average being 82·2 cmtr. (32·36 inches), and from 89·4—93·3 cmtr. (35·19—36·74 inches) during inspiration, the average being 90·7 cmtr. (35·71 inches); the maximum range of the inspiratory movements was thus 8·5 cmtr. (3·35 inches). The circumference of the upper part of the chest in women is about 76 cmtr. (29·92 inches), that of the lower part 70 cmtr. (27·55 inches).

A knowledge of the circumference of the thorax throws but little light on the condition of the internal organs; we learn merely that the chest is weakly or powerfully built. It is a well-established fact that a perfectly healthy state of the lungs is quite compatible with a chest-circumference of 78 cmtr. (30·7 inches) or even less.

The *diameters* of the thorax are, 1, the *long diameter*, measured from the clavicle to the base of the chest; 2, the *transverse diameter* (*the breadth*), a line drawn from a given point on one side of the chest to a corresponding spot on the other side; 3, *the antero-posterior diameter* (*the depth*), a line passing from any part on the anterior surface to a corresponding point posteriorly, most usually taken from the sternum to the vertebral column, whence the term *sterno-vertebral* diameter. There are various other special comparative measurements which are sometimes made on the two sides of the chest, such as the depth at the apex, from the clavicle to the spine of the scapula, the distance between the sternum and the nipples, or between the nipples and vertebral column, &c. The changes in the diameters of the thorax occasioned by diseases of the respiratory organs have already been stated (p. 29 *et seq.*). The long diameter is very variable, so much so that it is scarcely possible to fix on any one number as expressing its normal length; the transverse diameter in the upper and lower parts of the chest in adult men amounts generally to about 25—26 cmtr. (9·84—10·23 inches), in women to 23—24 cmtr. (9·05—9·44 inches), and to 1 cmtr. (0·39 inches) more a little above the level of the mamma; the antero-posterior diameter is about 16 cmtr. (6·29 inches) superiorly, 19 cmtr. (7·48 inches) in the middle and inferiorly.

When the chest is very much misshapen, especially from spinal curvature, the tape, as it cannot be accurately applied to the various parts, gives no useful or reliable information. In such cases Woillez' cyrtometer should be used; this instrument follows closely all the heights and depressions on the surface of the chest, and when removed furnishes us with an exact tracing of its circumference at the part examined.

This cyrtometer consists of a number of small rods of whalebone 1½ cmtr. in length so united by stiffly-moving joints as to form a non-resilient chain; at two points, coloured white, are hinges which are more freely movable than the others. The apparatus is to be applied closely to the chest and by pressure made to adapt itself to its form, dipping into its various depressions (intercostal spaces), &c.; it is then carefully removed, and its outline, drawn on paper, represents accurately that of the part of the chest examined. This measurement, repeated from time to time, affords valuable indications as to the progress of the disease under observation.

THE MOVEMENTS OF RESPIRATION.

In ordinary circumstances the dilatation of the thorax in respiration is effected simply by the action of the diaphragm and of the intercostal muscles, aided, in women, by that of the scaleni. In men the diaphragm is the most important of these agents; when relaxed it projects into the thoracic cavity in the form of a dome, but when contracted it becomes flattened and descends, pushing before it the abdominal organs, elevating the abdominal walls, and forcing outwards the cartilaginous parts of the lower ribs. This is termed, for obvious reasons, the *costo-abdominal type of respiration.* In women the enlargement of the chest takes place chiefly in the upper part (*costal type of respiration*), and is produced principally by the action of the intercostal muscles, and to but a slight extent by the contraction of the diaphragm. This type of respiration is not, as some hold, the result of embarrassment of the action of the diaphragm by tight lacing and the pressure of the various parts of a lady's attire, as it is observed in children of both sexes, who certainly are not exposed to these influences; it seems to originate rather in the greater flexibility of the ribs in both sexes during childhood, and in the female sex during the whole of life, the action of the intercostal muscles being more effective under these conditions. When respiration is very full and deep, in old age, and in certain pathological states, it occasionally takes the costo-abdominal form, even in women. All the diameters of the chest are increased in inspiration, the transverse and antero-posterior diameters by the movements of the ribs and sternum, the long diameter by the contraction of the diaphragm; it is by the latter means that the capacity of the chest is most of all increased.

The movements executed by the ribs are of two kinds, elevation and rotation. The *anterior* extremity of each rib is *raised* and *carried forward*, its fixed point being at the vertebral column; the rib is at the same time *rotated*, so that its convexity, which was before turned downwards, is now directed upwards and outwards. Further, as the ribs have a general inclination downwards and forwards from the spinal column to the sternum the effect of elevating them is to bring them more towards the horizontal position; this is clearly seen in deep inspiration and in attacks of dyspnœa. The sternum is raised and carried forward by the movements of the ribs. The antero-posterior diameter of the thorax is increased by the simultaneous forward and upward movement of the ribs, its transverse diameter by their rotation.

The lung follows every movement of the chest-wall. Its expansion in inspiration, as has been proved by exposing the pleura, and by carrying on artificial respiration after opening the thorax, takes place in two directions, from above downwards and from behind forwards; for the former movement the fixed point is the apex, for the latter a point on the posterior surface of the lung. If the lung at any part does not immediately expand when the thoracic cavity dilates—a common enough pathological phenomenon—this is at once indicated by the occurrence of a corresponding *depression* on the surface.

The diminution in the capacity of the chest in *expiration* is due solely to the relaxation of the inspiratory muscles and to the elasticity of the lung; the ribs and sternum return to their former position, and the distended lung contracts by virtue of its elasticity and expels the air which it contains.

The extent of the movement executed by the several parts of the thorax, which in normal and quiet respiration should be equal on both sides, is usually measured by passing an ordinary tape round the chest about the level of the nipples; in adults it amounts to 7—8·5 cmtr. (2·75—3·34 inches) when standing upright, to ½ cmtr. less when sitting. The changes in the transverse and antero-posterior diameters are indicated on the scale of the calipers. Inequality in the range of the movements on the two sides, even when slight, is usually readily detected, and points to the existence of some obstacle to the respiration on the side which lags behind, especially to such impediment as arises when a portion or the whole of one lung is completely collapsed or partially deprived of air. In the former case the corresponding side is generally motionless, or moves but little, in the latter case it expands, but not so freely as the sound side. These points may be observed even when the patient is breathing quietly; a full inspiration, however, brings them out more clearly, as the difference in extent of the respiratory movements in superficial and deep respiration is more marked on the healthy than on the affected side. Minute differences may often be recognised by examining the chest in profile, frequently also by watching the movements of the shoulder-blades. Thus, in patients suffering from large pleuritic effusion, when standing with the arms hanging downwards, the scapula on the diseased side is almost motionless, while that of the other side is considerably raised and has its lower angle turned forwards. If respiration be embarrassed in both lungs, as in bilateral emphysema, neither side of the chest moves much, sometimes not more than 5—6 cmtr., or even less. Should the obstruction be situated in the upper lobe of one or both lungs,—which occurs most frequently in phthisis pulmonalis,—the limitation of the respiratory movements is confined to the upper part of the chest on one or both sides. In the latter case the finer degrees of divergence from the normal range of movement are recognised with some difficulty, as we have no longer the advantage of being able to compare one side with the other.

Sometimes certain parts of the surface of the thorax near the apex on one side, more rarely on both sides, are observed to *sink* markedly *in inspiration* and to bulge outwards again in expiration. On close examination it is usually found that those parts, which are, as a rule, situated on the front of the chest and between the first and third ribs, are, even in the respiratory pause, somewhat less prominent than those in their immediate neighbourhood. These depressions are generally connected with condensation and the formation of one or more cavities in the subjacent lung tissue, and arise from the inability of the dense inexpansible lung parenchyma to follow the chest-wall when it is raised in inspiration, and the consequent yielding of the corresponding parts of the surface to the external atmospheric pressure.

Inspiratory depressions are also very common in the lower *lateral* intercostal spaces on *both sides*, in the epigastrium, in the region around the ensiform process and sternal insertions of the lower ribs, in the supra-clavicular regions and the suprasternal notch. These phenomena accompany the more severe forms of vesicular emphysema of the lungs and stenosis of the larynx (from croup, &c.). The cause in both cases is the rarefaction of the air within the lungs, and the consequent preponderance of the external over the internal atmospheric pressure. Direct proof of the accuracy of this explanation is found in the facts that inhalation of rarefied air from a pneumatic apparatus produces sinking of the supra-clavicular regions and of the flexible parts at the base of the chest, and that those concavities, when due to emphysema or stenosis of the larynx, disappear on the inhalation of condensed air.

It is evident that the air in the lungs must become rarefied in severe emphysema and in constriction of the larynx or trachea, as, notwithstanding the dilatation of the thorax and the expansion of the lungs produced by the energetic contraction of the inspiratory muscles, very little air enters the chest; the internal pressure is therefore abnormally lowered, the external pressure becomes the greater, and the most yielding parts and those most distant from the larynx are forced inwards. The diaphragm is quite unable to overcome this pressure from without, and is rather driven upwards by it. The *rigid* parts of the chest, as they offer sufficient resistance to the weight of the atmosphere, are not subject to these inspiratory depressions. These symptoms are much intensified on taking a deep inspiration, and are more observable in those who have wide intercostal spaces and are of spare habit.

In many cases of dyspnœa of old standing, especially in aged emaciated persons suffering from emphysema, a shallow horizontal sulcus (Harrison's Sulcus) marks externally the lower border of that portion of the diaphragm which rises from the xiphoid cartilage.

An important diagnostic point is often found in the relative extent of the movements of respiration in the upper and lower segments of the chest. When the impediment to the free entrance of air into the upper lobes is considerable the lower lobes act more vigorously and exercise vicarious functions, the diaphragm contracts powerfully, the lower part of the thorax is widely distended, while the movements in the upper parts are restricted. This type of respiration is often seen in consumptives. On the other hand, if the action of the diaphragm be more limited, as is often the case when it is carried upwards by the pressure of fluid or tumour in the abdominal cavity, or is forced downwards by pleuritic effusion or pneumothorax, the lower part of the chest-wall takes a less prominent share in the movements of respiration. Besides being hampered in its action from these mechanical causes the diaphragm may be partially paralysed from the persistence of the pressure and from the spread of the inflammation to its serous covering. In acute fevers having a protracted course (such as typhus) there is usually a temporary enfeeblement of the diaphragm.

More or less complete paralysis of the diaphragm from neuropathic causes is rare, and is distinguished by the position of the diaphragm (which is pushed upwards into the thorax), and by the passive retraction of the epigastrium and of those parts of the chest-walls from which the diaphragm springs. The appearances are thus the same, though not so well marked, as those which follow section of the phrenic nerve in animals, the diaphragm being passively drawn up into the chest at each inspiration and forced downwards into the abdomen at each expiration. In one case of partial paralysis, long under my own care, occurring in a man otherwise healthy, the bulging of the surface of the abdomen was wanting in shallow inspiration; with deep inspiration there appeared in the upper part of the abdomen a series of wavy elevations following closely on each other which, on expiration, returned and disappeared in the same order. These peculiar phenomena had lasted many months.

Stethography.

(The graphic representation of the movements of respiration.)

Various instruments have been invented for the purpose of graphically registering the movements of respiration. Vierordt and Ludwig's consists of a two-armed lever, the end of the shorter limb of which rests on the abdominal surface of the diaphragm, whilst the longer limb, fitted with a pencil, records its movements on a slip of paper which is caused to pass before it. In other apparatuses (Rosenthal's, Gerhardt's, Marey's, Fick's, &c.), though they differ much in form, the principle remains the same,—an arrangement of levers gives a tracing of the extent of the movements, on a plate which glides past the body at a certain rate. It is obvious that with these instruments the movements of but one spot can be noted at one time. Riegel, however, has constructed a "*double stethograph*" which enables us to examine at the same instant any two points on the surface of the chest, no matter how far apart, and thus to compare their tracings directly with each other. It consists of a strip of paper which is carried forward horizontally by clockwork, of two pencils which are kept applied to the two sides of the paper by means of lever-work, and of two separate levers which are set in motion by the rising and falling of the walls of the chest, and which communicate their oscillations to the pens. The tracings from the two sides of the chest, being on the same piece of paper, can be easily compared with each other. The tracings may be increased in size by lengthening the arm of one of the levers; in this way the slightest action of any part of the thorax may be demonstrated. The whole instrument rests on an iron stand, on the longer beam of which it is mounted and on which it is movable; the shorter beam bears the counter-weight. A simpler instrument than Riegel's has lately been devised by Haenisch; it, however, does not indicate all the variations in the respiratory movements, but only the length of the inspiratory excursion at two corresponding points, such as the apices.

Investigations carried on by means of the double stethograph show that under normal conditions inspiration is generally somewhat shorter than expiration, and that between them there is no pause,—that inspiration, having reached its termination, is at once, though almost insensibly, followed by expiration; and further, that the pause between any two respirations is exceedingly short. All parts of the thorax are not engaged in the same stage of the respiratory act at a given moment; inspiration may be completed at one point but only begun at another, or in one part expiration may be only ending when in another inspiration has already commenced. There may be absolutely no quiescent interval when respiration is much accelerated; on the other hand, it is well marked when the breathing is slow. A comparative examination of the tracings also shows that in men both sides of the chest, all the intercostal muscles, and the diaphragm, are called into action simultaneously, and that the inspiratory elevation is equal on both sides; that in young persons not unfrequently the upper part of the thorax moves more than the lower (a condition which is reversed in old age); and that in women the range of movement of the parts of the thorax decreases from above downwards.

When pathological changes take place in the respiratory apparatus the stethograph gives much the same indications as simple inspection, but usually of a more striking character; thus the slight degree of elevation shown in the inspiratory tracing of a partially or completely collapsed lung presents a marked contrast to the high curve of the sound lung. Obviously the tracings are not diagnostic of the various causes of collapse of the lung (pleuritic exudation, pneumothorax, pneumonia, phthisis, &c.), as they show merely that the respiratory movements are restricted; the greater the collapse the deeper the curve. Parts which sink in inspiration and become prominent in expiration give a reversed tracing, the inspiratory line descending the expiratory line ascending. The most characteristic tracings are those of stenosis of the larynx and trachea, and of emphysema of the lungs. In the first the expiratory curve is normal, while the inspiratory curve is much prolonged, corresponding to the long duration of inspiration produced by the blocking up of the air-passages; in emphysema it is the expiratory line which is the longer, as it is the escape of air from the chest which is most impeded. In severe and extensive emphysema the expiration is somewhat irregular, being relatively rapid in its first two-thirds; the obstacle to the expulsion of the air is then suddenly encountered, when, in spite of the powerful action of the respiratory muscles, the rest of the expiration is accomplished slowly and with difficulty. This is all very clearly shown in the tracing, the accession of the hindrance to expiration being indicated by a sharp angle. In chronic catarrh this phenomenon is quite absent, expiration being uninterrupted; it is therefore graphically distinguishable from emphysema.

FREQUENCY OF THE RESPIRATION.

This, in health, amounts to 14—18 per minute in adult men, to a somewhat larger number in women and children, and to 40 or more in the new-born. One respiration usually corresponds to four beats of the pulse. Position has but little influence on the frequency of the respiration, though it is rather faster when sitting or standing than when lying. The most extensive control is exercised over it by the will,—it may be voluntarily deepened or made superficial, accelerated or retarded, or even arrested for 30—60 seconds. It goes on most quietly and regularly when Will and Perception are in abeyance, as in sleep.

Of the deviations from the normal frequency acceleration is more common than retardation. The rate may increase to 70, 80, or even to over 100 per minute; generally, however, it rises no higher than 40. Abnormal rapidity of respiration is called *dyspnœa*. Respiration is sometimes not merely quickened, but each inspiration may gain considerably in depth; at other times respiration is simply fuller, while its speed is scarcely increased, or may even be diminished. The latter condition also is known as dyspnœa.

Respiration is physiologically accelerated by physical exertion, such as rapid walking, running upstairs, and generally by all those circumstances which tend to increase the action of the heart. Those who are convalescent from severe acute diseases, therefore, begin to breathe more quickly as soon even as they sit up in bed, as the heart is thereby excited to more powerful and rapid contraction; the same effect is produced in some patients by mental depression or even by the knowledge of the fact that they are being observed by others. All these disturbing influences have to be carefully weighed or excluded when we wish to determine the presence or absence of abnormal frequency of respiration, a symptom which, even when but slightly developed, has always great pathological significance.

Acceleration of the Respiration takes place as a *pathological phenomenon* in the following circumstances—

1. When there is *pain* in any part of the thorax or abdomen to which the movements of respiration are communicated. The patient breathes hurriedly but superficially, in order to avoid adding to his suffering by causing disturbance of the parts affected,—a condition of which the onset of pleurisy affords a good example. Painful affections of the ribs and of the muscles of the thorax (as in acute muscular rheumatism) are, in the same way,

associated with increased frequency of respiration, to but a slight extent, however, when the pain is located in the investing (pectoral and dorsal) muscles, most markedly when the proper respiratory muscles (the intercostals and diaphragm) are implicated.

In one case of acute rheumatism of the entire muscular system of the chest I found that the respirations numbered 40 per minute; in another case, in which the symptoms seemed to point to inflammation of the diaphragm, the respiration was over 50 per minute, while the diaphragm remained almost motionless, the slightest attempt at deep inspiration being cut short by agonising pain.

The breathing of those who are suffering from acute pain in the abdomen (as in diffuse peritonitis) is exceedingly shallow, and therefore very rapid, as the contraction and downward movement of the diaphragm and the consequent displacement of the abdominal organs are thus reduced to a minimum.

2. Increased frequency of respiration is an unfailing concomitant symptom in all *febrile conditions* of any considerable degree of intensity, from whatever cause they may arise; it does not, however, increase proportionately to the severity of the fever. In very high fever the number per minute may reach 30—40, in children 50—60, without necessarily implying the existence of any lung complication. This is attributable to various causes,—to the acceleration of the pulse and the greater velocity with which the blood circulates through the lungs, to the augmentation of the quantity of carbonic acid contained in the blood, but most of all to the abnormal elevation of temperature. Through those influences the respiratory centre in the medulla oblongata is more often and more powerfully stimulated and the reflex movements of respiration are more frequently excited.

That the rapidity of the breathing in fever depends more on the temperature of the blood than on the rate of the pulse is proved by the facts that many cases occur in which a high degree of heat and a relatively slow pulse are associated with acceleration of the respiration, and that, on the other hand, there are cases in which respiration becomes gradually slower as the temperature is reduced by the use of cold baths, the pulse-rate remaining unchanged. Dyspnœa from this cause (*heat dyspnœa*) is the result of direct irritation of the respiratory centre, as has been shown by numerous experiments on animals, in which the blood, as it passed through the carotids, was artificially warmed.

3. The most marked increase in the frequency of the breathing takes place in those diseases in which *the interchange of gases* that should go on *in the lungs is disturbed,* diseases whose seat may be either in the respiratory organs themselves or in other parts. In the former case the dyspnœa arises from the blocking up of some of the avenues by which the air reaches the breathing surface, either by *diminution of the calibre of the air-passages* or by *conditions which render the pulmonary vesicles impermeable.*

The cases in which the air-canals are *narrowed* may be divided into two classes, those in which the obstruction is situated in the larynx or trachea, and those in which it occurs in the larger bronchi or their finer subdivisions. To the former group belong œdema glottidis, croup, diphtheritis of the larynx, and thyroidal tumours which compress the trachea ; the latter group includes especially catarrhal swelling of the bronchial mucous membrane, which, when it is extreme, affecting a large number of the finest air-tubes, produces all the effects of constriction higher up—in the larynx or trachea.

The *alveoli* may become *impervious* from *infiltration* with plastic or fluid exudation (in pneumothorax, cheesy degeneration, œdema of the lungs), or from compression (by fluid, air, tumours in the pleura, &c.), or from loss of elasticity to such an extent that they do not sufficiently expand in inspiration nor adequately contract in expiration (as in vesicular emphysema). When, from one or more of the above-named causes, the air-cells are closed the decarbonisation of the blood is interfered with, the blood becomes deficient in oxygen and contains an excess of carbonic acid, and the respiratory centres are more frequently and more strongly excited to action. The degree of the dyspnœa depends on the more or less complete occlusion of the alveoli and on the area involved in the changes which lead to this result ; with certain reservations it may be said that, other conditions being the same, it increases with the magnitude of the obstacle to respiration, that is, with the diminution of the available breathing surface. As a rule it is the more marked the more suddenly the respiratory surface is reduced ; thus, in collapse of one lung from pneumothorax, developed within a few hours, it reaches a high degree of intensity ; but should the collapse be brought about by the pressure of a slowly-accumulating pleuritic effusion, the dyspnœa is less considerable.

When, as in pleuro-pneumonia, pain and fever co-operate with the cause just described in accelerating the respiration, the rate shows a very rapid increase and may soon rise to 40 or more per minute; on the other hand, certain varieties of chronic consolidation of the lungs, inasmuch as their course is painless and free of fever, are accompanied by much less disturbance of the respiration than pneumonia, even when they disable a larger portion of the lung. I have seen cases of complete atelectasis of an entire lung, produced by pleural effusion of long standing, in which the respirations were not more than 24 per minute nor appreciably increased in depth.

In chronic affections in which respiration is obstructed its frequency varies greatly, particularly according as the body is well or ill nourished. Thus in phthisis pulmonalis, even when it is far advanced and the breathing surface extensively destroyed, respiration is often not at all hurried, because, on account of the emaciation, *the quantity of blood in circulation is decreased* and a much smaller volume of oxygen suffices for its decarbonisation. In those destructive processes, however, which are not attended by wasting, such as bronchiectasis, breathing is usually greatly accelerated. Respiration becomes exceedingly rapid also when a new obstacle is suddenly added to one of older standing. Emphysematous patients feel tolerably comfortable as long as their shortness of breath is due merely to inexpansibility of the air cells; but should a diffuse bronchial catarrh supervene or should the original disorder be seriously aggravated by cold weather, their dyspnœa immediately becomes very distressing.

The frequency of respiration is further increased by certain *diseases of the heart*, especially by those *valvular affections* which give rise to *overfilling of the pulmonary circulation* and thus to interruption of the free interchange of gases in the pulmonary capillaries. Among such conditions are insufficiency of the mitral valve and stenosis of the mitral orifice; in the first case a quantity of the blood in the left ventricle passes backwards into the auricle at each systole, in the second the free flow of blood from the left auricle is prevented. Under these circumstances the left auricle, and subsequently the pulmonary capillaries and veins, become engorged, the necessary consequence of which is that the blood is insufficiently arterialised in the lungs, the respiratory centre is more powerfully stimulated, and the movements of respiration more frequently repeated. A second, but less important, element in the production of dyspnœa in mitral disease is the occurrence of bronchial catarrh from the passive congestion of the circulation through the lungs. Those heart affections in which the pulmonary circulation is intact, such as diseases of the aortic valves, usually produce at first no dyspnœa; but in their later stages, when the left ventricle undergoes fatty degeneration and is no longer able to propel the whole of its contents into the aorta, and so fails to accommodate the whole of the blood which should enter it from the left auricle, the pulmonary vessels are overloaded and dyspnœa is established.

Slight dyspnœa, however, is set up in aortic disease, from the increased rapidity of the heart's contraction, and when the hypertrophied and dilated left ventricle encroaches on the neighbouring parts of the lung, causing them to shrink and so diminishing their respiratory surface. The dyspnœa attendant on large aneurisms of the aorta may be explained in the same way, though in this case it often arises from pressure on the trachea. Disease of the tricuspid valve or of the valves of the pulmonary artery very seldom occurs alone, but is usually combined with aortic or mitral disease, so that dyspnœa in these cases is of a somewhat complex origin.

Increased rapidity of the respiration may be due to diseases of the abdominal organs in which the abdominal cavity is distended, the diaphragm being pressed upwards into the chest and its movements in this way limited. Dyspnœa is thus frequently observed in cases of ovarian tumour, ascites, and tympanites, especially when the patient assumes the dorsal position. The greater the number of these exciting causes which coexist in any given case the more marked is the dyspnœa; it takes probably its most agonising form in the later stages of mitral disease, when ascites, hydrothorax, and pericardial effusion make their appearance together.

Not unfrequently we have dyspnœa proceeding from no obvious cause, or at least from no cause which corresponds to it in apparent importance. In most of these cases it is owing to a temporary diminution in the calibre of the bronchi, from spasmodic contraction of the bronchial muscles; it is therefore named *bronchial asthma*. This variety of dyspnœa occurs in paroxysms of relatively short duration, while in the intervals, when the case is not complicated with other affections, especially bronchial catarrh, respiration may be almost normal.

The theory that these dyspnœal attacks are referable to bronchial spasm is well supported by experimental evidence. Irritation of the vagus nerves in the neck is followed by *contraction of the unstriped bronchial muscles*, and therefore by diminution of the lumen of the bronchi; its effect also on the contractility of the lungs is indicated by the rising of a column of fluid in the tube of a water manometer attached to the trachea (Donders, Bert, Gerlach, &c.). It is especially the finer and smallest air-tubes which take part in this spasm, though it is not impossible that the larger bronchi may likewise be affected, as the trachea of animals has been shown to contract on electrical stimulation (Horwath). It has not yet been definitely ascertained whether the alveoli themselves also shrink on irritation of the vagus. The bronchial catarrh which almost always accompanies asthma has manifestly but an inconsiderable influence in causing the dyspnœa, as the latter occurs in paroxysms which, when they subside, are succeeded by an interval in which the breathing is easy and natural, this remission being altogether independent of any change in the catarrhal symptoms. In other cases, more rare than those just described, dyspnœa may be periodical and of great severity, while not the slightest morbid alteration may be found in the respiratory or circulatory organs. It may be assumed that these phenomena are caused by some transient irritation of the vagi, though the particular circumstances in which this irritation is excited are still unknown. In the case of a young girl, whose internal organs were perfectly healthy, I have seen the respiration become as rapid as 40 per minute, and sink again in a few days to the normal rate. For many years I have had under observation another case, that of a woman, whose organs of respiration and circulation have remained perfectly intact, but who is subject to extremely severe paroxysmal attacks of dyspnœa, accompanied by a feeling of intense anxiety, during which the frequency of the respiration increases to 70, 80, sometimes almost to 100 per minute; the violence of the seizure then gradually abates, and respiration again becomes slow and quiet. These attacks sometimes take place daily, at other times they are absent for months together. Bischoff has recorded a somewhat similar case; the breathing, during the paroxysms, was greatly increased in rapidity, sometimes to 160 per minute, but in the intervals was perfectly normal.

From *objective* dyspnœa is to be distinguished a *subjective* variety. Many persons complain of occasional shortness of breath, especially those who have a sense of weight or pain in the epigastrium from diseases of the stomach or from other causes, those who have a feeling of oppression in the cardiac region, hysterical individuals and others, in whom no trace of any physical condition which might explain this subjective dyspnœa can be detected. While it lasts no dyspnœa is objectively apparent, or the patients, while breathing superficially but at the normal rate, only now and then draw a full, deep inspiration,—a state of matters very frequently met with even in perfect health.

Genuine dyspnœa is distinguished by another peculiarity, namely the *greater depth* and therefore the *longer duration*, of each respiratory act. As a rule the depth is inversely proportionate to the frequency. Thus, in the severe acute diseases of the respiratory organs (such as pneumonia) the breathing is rapid but not appreciably increased in depth,—the respiratory muscles being evidently unequal to a task demanding so much exertion, as, in common with all the muscles of the body, they participate in the general emaciation caused by the high fever. On the other hand, in those chronic lung diseases in which the physical strength is not reduced,—vesicular emphysema for instance,—respiration is often very deep and full while the increase in its frequency is but slight. There are *two forms* in which this greater depth or longer duration of respiration may manifest itself,—it may affect principally the *inspiration* or the *expiration*. Prolonged inspiration, *inspiratory dyspnœa*, is observed when the air enters the lungs with difficulty, as in stenosis of the larynx and trachea; its most typical and simple form is seen in paralysis of the posterior crico-arytenoid muscles, the margins of the glottis in that rather rare affection being approximated to each other in inspiration and relaxed in expiration,—the reverse of the normal movements. Prolonged expiration, *expiratory dyspnœa*, occurs when the escape of air from the lungs is in any way impeded, as in diffuse bronchial catarrh associated with vesicular emphysema, though in these circumstances the inspiration also is lengthened.

The greater depth of the respiration is the result of the increased action of the ordinary respiratory muscles assisted by that of the *accessory* muscles, the latter term including those which, though they take no part in the thoracic movements when the breathing is normal and quiet, contract powerfully when it is desired to expand the chest to the utmost.

The accessory inspiratory muscles in the neck are the *scaleni* and the *sternomastoids*. The anterior and middle scaleni raise the first rib, the posterior scalenus the second; these, however, especially in the female sex, are not entirely inactive even in ordinary respiration. When the head is fixed the sternomastoids elevate those parts into which they are inserted,—the clavicles and sternum. Certain of the thoracic muscles also may be considered accessory muscles of inspiration.

The *pectorales* (major and minor), when the dyspnœa is very intense, raise the ribs (second to sixth) when the head and shoulders are fixed; it is by availing themselves of the action of these muscles that emphysematous patients, when suffering from an attack of asthma, seek to obtain relief in grasping firmly some object above their heads. The *serrati postici superiores* elevate the upper ribs; the *subclavius* has a similar action on the first rib when the clavicle is stationary; the *levatores costarum* (breves and longi) draw the posterior portion of each rib towards the vertebral column; the *levator anguli scapulæ*, that part of the *trapezius* which rises from the occiput and is inserted into the clavicle and acromion, and probably also the *serrati antici majores*, act as inspiratory muscles, inasmuch as they move the lower and middle ribs upwards and outwards when the shoulder is fixed. Finally, it has been proved by experiments on animals that in asphyxia the elevators of the head and spinal column aid in inspiration; in men it is only when suffocation is threatening, in croup and spasm of the glottis, that these muscles are called into action.*

While the above-mentioned muscles are to be regarded as *dilators of the thorax*, others, such as the *levatores alæ nasi* and the *levator palati mollis*, co-operate with them by enlarging the openings by which the air enters; the *sterno-hyoid*, *sterno-thyroid*, *thyro-hyoid* and *omo-hyoid* muscles also, by depressing the larynx, facilitate the admission of air into the lungs. The most important of the accessory muscles of this kind are the *crico-arytenoidei postici*; when respiration is at all embarrassed they at once contract powerfully, separate the arytenoid cartilages, and so dilate the rima glottidis.

In animals the various groups of accessory muscles of inspiration are brought into play in a certain order, according as the difficulty of breathing increases; thus Traube states that first the elevators of the upper ribs are thrown into violent contraction, then the scaleni, the elevators of the lower ribs, the sterno-hyoids and sterno-thyroids, and lastly, the serrati postici superiores. In the human subject no such order is observed, the additional movement is sometimes accomplished by one set of muscles, at other times by another. In general, however, when the dyspnœa is considerable, the cervical muscles (especially the scaleni and sternomastoids) are the first to come to the aid of the ordinary inspiratory muscles; when the dyspnœa is still more severe these are joined by the levatores alæ nasi, and in the worst cases by the muscles of the breast and shoulder-blade.

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* One may easily, by simply fixing the arms, demonstrate on his own person the fact that these muscles are really instrumental in adding somewhat to the forces which dilate the chest

Expiration, even when abnormally prolonged by the presence of diffuse bronchial catarrh or other obstacle to the emptying of the lungs, is usually effected by the elasticity of the lungs alone. When this elasticity, however, is much diminished, as in the advanced stages of emphysema, it is supplemented by the action of various muscles; the thorax is thereby as far as possible reduced in volume, and so made capable of greater expansion at next inspiration. The principal *expiratory muscles* are those of the abdomen; they compress the abdominal organs and thrust them upwards towards the diaphragm, the transverse diameter of the abdomen being shortened by the action of the *transversalis*, the long diameter by that of the *recti*. Other muscles assist in expiration by drawing the thorax downwards; the *obliqui* (external and internal) and the *triangularis* sterni depress the anterior part of the lower ribs, the *serrati postici inferiores* (antagonists of the serrati postici superiores) have a similar influence on the four lower ribs, and the *quadratus lumborum* on the lowest rib.

The existence of some difficulty of breathing may frequently be recognised at the first glance by the marked contraction of several of the accessory muscles of inspiration, such as the sternomastoid, scalenus, omohyoid, and upper part of the trapezius. Should these muscles become hypertrophied by frequent exercise of their function, as often happens in patients who have long suffered from emphysema, each contraction causes them to stand out prominently in their whole length, a phenomenon which is exceedingly striking when the neck is thin and emaciated, and when the supra-clavicular regions sink at each inspiration. The contractions of the accessory expiratory muscles also are sometimes visible.

The effective operation of these accessory muscles depends essentially on the *position* of the patient. When decubitus is dorsal the muscles of the back, when lateral those of the undermost side, act but feebly; the patient therefore instinctively assumes such a posture as is best adapted for the vigorous contraction of the most powerful of the inspiratory muscles or which permits of the co-operation of the greatest number of them. Accordingly the position of the dyspnœtic patient,—excepting those cases in which he is not perfectly conscious, and does not therefore select the most comfortable attitude,—furnishes us with a useful indication of the greater or less gravity of the impediment to the respiration. Thus, the obstacle cannot be one of any very great magnitude if the patient habitually lies on his back; with every aggravation of the difficulty of breathing he assumes a higher position, and ultimately has to remain almost constantly in the sitting posture (*orthopnœa*). In the most intense forms of dyspnœa, such as occur in the asthmatic attacks connected with old-standing emphysema, in diseases of the heart associated with ascites, hydrothorax, &c., the patient is sometimes unable to sit in bed, but is obliged to pass many sleepless nights in a half-standing position or in an arm-chair.

The semi-erect posture is often adopted even when the interference with the process of respiration is not of a very serious character, especially if the affection of the lungs or air-passages is attended by a profuse fluid secretion; in this position the accumulated matters are more easily expelled on account of the greater assistance which the muscles of expiration are then able to render.

The exact seat of the obstacle to respiration has also considerable influence in determining the habitual attitude of the patient. If it involves *one side* of the chest only, as in pleurisy with effusion, it is found that breathing is more easily performed when he lies on the *affected* side, as the muscles of the sound side are thus left completely unencumbered, and the healthy lung is at liberty to dilate to its fullest extent and so to partially compensate for the disablement of the other lung. Should the patient turn to the sound side the dyspnœa is at once greatly augmented, for not merely does the hindrance to muscular contraction on the affected side continue as before, but on the sound side also the action of the chest is hampered, and the weight of the fluid pressing on the healthy lung still further reduces its available respiratory surface. On the other hand, at the *commencement* of painful unilateral pleurisy the patient generally elects to lie on the unaffected side, as resting on the diseased side increases the pain. For the same reason when there is a painful condition of *one side* of the chest arising from other causes decubitus is usually on the sound side. These observations regarding decubitus apply only to those diseases which are characterised by the severer forms of dyspnœa; in milder cases the patient reclines indifferently on either side, according to habit.

When the function of respiration is in any way impeded, increase in frequency and in depth are the most common changes that the act of breathing undergoes. But in some rarer cases respiration is not at all accelerated, or is even somewhat *slower* than in health; inspiration, however, is then always prolonged and much more full. This type of respiration is observed in two conditions:—

1. In stenosis of the larynx or trachea, acute or chronic.
2. In lung diseases which are followed by affection of the brain.

The first condition is best illustrated by laryngeal croup—in children the most frequent cause of laryngeal stenosis; in it inspiration is prolonged, slow, and deep, and accompanied by a peculiar crowing noise (*stridor*) which is audible even at some distance. This retardation of the respiration is due to the *constriction of the rima glottidis* which must always occur on the deposit of croupous exudation above and below the glottis, but which may also take place independently of the presence of the croupous membrane, from inflammatory swelling and consequent partial paralysis (paresis) of the muscles which govern the vocal cords, especially those which relax them. It is thus only by dint of very violent effort on the part of the dilators of the glottis that the passage of air is permitted, so that *inspiration becomes laborious and deep;* superficial, though frequent, respiration would be of little avail under such circumstances. Further, in those cases in which the vocal cords are covered with exudation *expiration* also is obstructed and prolonged; and as both inspiration and expiration thus occupy a longer period respiration as a whole is slower. Section of the recurrent laryngeal nerve in animals produces narrowing of the glottis and gives rise to the same phenomena—diminution in the frequency of respiration and deepening of each individual inspiration.

This explanation of the cause of retardation of the respiration is applicable to most cases of stenosis of the larynx, even to those in which expiration is *not* obstructed. Thus, if a patient suffering from paralysis of both posterior crico-arytenoid muscles, in which there is only *inspiratory* contraction of the glottis, should breathe rapidly he would be devoting as much time to expiration (which is free) as he does to inspiration (which is difficult); he instinctively prefers, therefore, to protract inspiration at the expense of expiration.

Experiments on animals furnish the true explanation of the slowness of respiration in the second variety of cases, those which are complicated by the occurrence of brain disease. It is well-known that the respiratory centre is situated in the medulla oblongata, close to the point of origin of the vagus nerves. It is through these nerves that the respiratory impulse is excited and the rythmical character of the movements sustained; when they are divided in the neck the frequency of respiration sinks considerably, sometimes to even one-third its normal rate per minute, while each individual inspiration becomes exceedingly deep and is accomplished only by great muscular exertion; and if the respiration of the animals so operated on be still further embarrassed, as by puncturing one side of the chest or by injecting fluid into the pleura till one lung is fully compressed, and the functionally active lung-surface in that way suddenly reduced by one-half, the breathing nevertheless remains slow and quiet as before. Similarly in men, respiration, notwithstanding the existence of a considerable obstacle to its proper performance, may be unduly retarded when the normal stimulating influence of the vagi is withdrawn, an accident which is apt to happen when their medullary roots and the adjacent parts of the brain are compressed by fluid exudation, extravasation of blood, and other changes in the basis cranii. This same symptom, prolonged and deep inspiration, is observed also in limited regional brain diseases [Heerderkrankungen] affecting even parts distant from the vagus,—in the coma which follows apoplectic seizures, and in circumscribed softening.

When the vagus is compressed by tumours in the neck, or wounded during operation or by gunshot, respiration becomes less rapid, though not to such a marked degree as when the nerve is divided in animals. I have elsewhere recorded a case of this nature, in which the respirations were 12 per minute and inspiration was abnormally deep, dependent on paralysis of the vagus from diphtheritis of the fauces.

There is a special form of dyspnœa, known as *Cheyne-Stokes' Respiration*, which is met with in some diseases of the heart and brain, and in certain other affections in the course of which cerebral complications arise. It consists in a regularly-occurring pause, lasting ¼—1 minute, during which respiration is completely suspended, and which is preceded by the following characteristic train of dyspnœal phenomena: inspiration, which is at first short and shallow, becomes gradually deeper, and finally markedly dyspnœal; when this dyspnœa has reached the acme of its severity respiration becomes again more and more superficial with each successive inspiration, and eventually comes to an absolute standstill. When the pause has continued ¼—½ minute these symptoms are repeated in exactly the same way. In the most pronounced cases the dyspnœal period, during which the respirations number about 30, occupies ½—¾ of a minute, and the pause is of nearly the same duration. The whole cycle of phenomena therefore takes up 1—1½ minutes; in less marked cases it lasts only about ¼ minute, and may easily be overlooked, especially when the pause is very short. This variety of dyspnœa rarely occurs in the early stages of any disease, usually appearing only a few weeks, days, or even hours before death; it is, therefore, with but few exceptions, a fatal indication.

Stokes' respiration is observed in a great many different affections, relatively most often in those diseases of the brain which give rise to compression in the neighbourhood of the medulla oblongata,—hæmorrhage, exudation, œdema, tumours, uræmia,—in certain cardiac diseases, such as fatty degeneration, sclerosis of the coronary arteries, stenosis of the aortic or mitral orifices, &c. As all these conditions have one common feature,—diminution of the arterial blood supply, and so of the supply of oxygen, to the brain or respiratory centre in the medulla oblongata,—Traube has assumed it as the starting point of his explanation of Stokes' phenomenon. His theory is that *the irritability of the respiratory centre is* so materially *lowered* by the inadequacy of the supply of oxygen that a much larger accumulation of carbonic acid in the blood than is usual is necessary for the excitation of the inspiratory impulse, that thus the time required for the taking up of the requisite quantity of carbonic acid from the tissues is longer, and that therefore the intervals between each inspiration are more protracted. In opposition to this hypothesis, Filehne, founding his views on some observations made on animals (which, when strongly narcotised, breathe in the above-described fashion) and on men, has shown that though the irritability of the central organ of respiration is indeed diminished, this alone is not sufficient to produce Stokes' respiration, but that it is further essential that it be less easily excited to action than the vasomotor centre. The symptoms are caused therefore in the following way: at the end of the respiratory pause there is a large disappearance of oxygen from the blood, carbonic acid has accumulated, the vasomotor centre is thereby stimulated, and the arteries (the cerebral arteries among the rest) at once contract; this produces a gradually-increasing anæmia of the respiratory centre, and inspiration becomes more and more deep; this, however, supplies the wanted oxygen to the blood, the arterial spasm is relieved, the anæmia of the respiratory centre passes off, and with it the exaggerated impulse to respiration, and breathing once more becomes superficial. When the arterial spasm has entirely subsided, so that the respiratory centre is abundantly provided with decarbonised blood, the stage of apnœa, the pause, is reached, and lasts till, by the abstraction of oxygen from the blood, the irritation of the vasomotor nervous centre is renewed and the whole series of operations again gone through. That the arteries are strongly contracted is proved by the increase of the arterial tension and of the blood-pressure, while the anæmic condition of the brain is demonstrated by the fact that in young children, at the end of each respiratory pause, immediately before the re-commencement of respiration, and also while inspiration is gaining in depth, the great fontanelle is depressed. And further, this form of dyspnœa may invariably be arrested at the very commencement of each seizure by the inhalation of nitrite of amyl, which dilates the vessels. We are thus able to produce experimentally in animals all the phenomena which go to form Stokes' type of respiration; we can diminish or even cut off the blood supply of the brain, and so set up a gradually-increasing dyspnœa, and we can then re-establish the circulation and in that way reduce the dyspnœa or even arrest respiration.

SPIROMETRY.

(The estimation of the vital capacity of the lungs.)

By the term *vital capacity* is understood the greatest volume of air that can be inhaled by the fullest inspiration after the most forcible expiration; it must not be confounded with the term *total capacity*, which means the vital capacity with the addition of that quantity of air which still remains in the lungs even after the most complete expiration. The latter, the residual air, is displaced only when the lungs collapse on opening the thorax.

The vital capacity of the lungs may be most conveniently measured by means of Hutchinson's *Spirometer*.

This instrument consists of two cylinders, the outer of which is open above and filled with water, on which the inner one floats: the inner cylinder is open at the lower end, and properly balanced by weights placed on its upper closed end. A piece of elastic tubing, through which the patient is directed to breathe, is connected with the larger outer cylinder. In expiration the inner cylinder rises, in inspiration it sinks, and the distance through which it moves is registered on a scale graduated to show the number of cubic centimeters of air inhaled or expelled. It is seldom that the apparatus is used for determining the volume of air taken into the lungs, inspiration from the cylinder through the tube being somewhat unpleasant. To measure the amount of expired air the receiver is lowered so as to cause the index to point to a low figure on the scale, the nostrils are closed, a deep inspiration is drawn, and the air driven from the lungs through the tube and into the cylinder; the latter is buoyed upwards by the air which enters it, and remains fixed on the completion of expiration. If, for example, the index has advanced on the scale from 2,000 ccm. to 5,000 ccm., the vital capacity of the individual is said to be 3,000 ccmtr.

The vital capacity of the lungs of healthy persons varies considerably,—from 3,000 to 4,500 or 5,000 ccm. in men, and from 2,000 to 3,000 ccm. in women. Besides sex the elements whose variation seems to influence it most are height and age. It increases at the rate of 60 ccm. for every centimeter of stature above 155; thus if the vital capacity which corresponds to a height of 162 cmtr. be 3,000 ccm., that associated with a height of 167 cmtr. should be 3,300 ccm. Its relation to age is seen in an increase of about 160 ccm. from the fifteenth to the thirty-fifth year, and a decrease of about 900 ccm. from the fortieth to the sixty-fifth year; it is further very low in the extremely aged and in young children. Sedentary occupation and deficient nutrition lessen, the opposite conditions augment, the vital capacity; it also undergoes diminution in the sitting posture, and when the stomach is distended with food. The average in men of 20—40 years of age and of medium height may be assumed to be 3,600 ccm., in women 2,500 ccm.; in shorter individuals it may be 3,000 or less, while in tall persons it may mount to 4,000 or more.

The vital capacity is *diminished* in all diseases of the respiratory organs in which the expansibility of the lungs is interfered with; in the later stages of such affections it may sink to half its normal amount, or even lower. This fall is dependent on, but is not exactly proportionate to, the extent of breathing surface encroached upon; thus, if half of the respiratory area is disabled, as by compression of one lung by pleuritic effusion, it does not follow that the vital capacity will be similarly lowered by one half, as under such circumstances the healthy lung expands more freely, and so compensates somewhat for the loss of function in the affected lung.

Like every method of examination which involves the use of large or unwieldy instruments spirometry has never come to be regularly employed as an aid to diagnosis in the diseases of the thoracic organs, more especially as the data so furnished are, as a rule, less trustworthy than those easily obtainable by other means. In those cases also in which physical examination alone is sufficient to make it perfectly clear that the capacity of the lungs is diminished,—as in pleurisy with copious effusion, advanced emphysema, &c.,—it is superfluous to demonstrate that fact further with the spirometer. Spirometry is thus most useful when the decrease in the vital capacity is so trifling as to escape detection by other tests,—as when infiltration is beginning in the apices of the lungs; even then, however, it is scarcely to be expected that so slight an organic change should very markedly hamper the breathing, as complete infiltration of one apex lowers the vital capacity by little more than 100 ccm. Further, the results may vary to the extent of 100 ccm., according as the operation is performed with due care and attention to all essential details or otherwise; even persons practised in the use of the apparatus do not always succeed in raising the index to the same point. The vital capacity also differs so widely in individuals of the same height, age, and sex that, unless the usual and normal capacity of the patient under examination be known, one is warranted in making pathological deductions from it with confidence only when it falls decidedly below the physiological minimum. Finally, when the full dilatation of the lungs is from any cause prevented (as by pain), when respiration is abnormally frequent, or when the patient is very weak, spirometry can scarcely be said to be available as a means of diagnosis.

Nevertheless comparative spirometric observation, carried on for some time and often repeated,—the above-named sources of error being excluded,—comes to be of considerable value, as indicating the progress of the disease (whether improving, stationary, or being further developed).

The vital capacity is *increased*, in health as well as in disease, by *residence in elevated regions* and by the use of certain *pneumatic apparatuses*,—by *inspiration of compressed air*, which produces greater expansion of the lungs, and by *expiration into rarefied air*, which permits of the more complete emptying of the lungs: in the latter case a portion of the residual air also is expelled, the total quantity of air displaced is greater than usual, so that the lungs are made to dilate more fully in the following inspiration.

The most widely-known pneumatic instrument is the portable apparatus introduced some years ago by Waldenburg, which is so constructed that the patient can at will inhale or expire into condensed or attenuated air. For therapeutical purposes the vital capacity may be augmented by expiration into a chamber the pressure within which is 1-60th, 1-40th, or 1-30th, less than that of the atmosphere, or by inspiration of air whose density is 1-60th—1-30th greater than that of the atmosphere. Inspiration of rarefied air does not add to the vital capacity, but it stimulates the inspiratory muscles, making them act more energetically, and in that way overcome the difficulty experienced in breathing in such circumstances. Expiration into compressed air does not affect the vital capacity, and is therefore not resorted to in the treatment of disease. Though the volume of air set in motion in each act of respiration may thus be increased either by expiration into rarefied air or by inspiration of condensed air, these two methods of accomplishing that object cannot be used indifferently. In pulmonary emphysema, for instance, in which the alveoli are abnormally distended and inelastic, taking almost no part in the movements necessary for the expulsion of air from the chest, the deficiency is chiefly in the expiration; here the *vital capacity should be increased by expiring into an atmosphere of less than the normal pressure*, as by this means the *contraction* of the vesicles themselves is favoured and a much larger volume of air (1,000—2,000 ccm. more) given out than in the most forcible expiration under ordinary conditions: inspiration of compressed air, however, would only have the effect of still further stretching the walls of the pulmonary vesicles and so of reducing the dyspnœa (for which purpose, indeed, it is sometimes practised) but not the emphysema. When, on the other hand, the lung has become partially consolidated by compression (as in cases of pleuritic effusion), in stenosis of the air-passages, and in phthisis, the lowering of the vital capacity is owing to the inability of certain parts of the lungs to expand sufficiently, so that the defect is principally in inspiration; here it is evident that the most appropriate method of treatment is to *promote the expansion of the lungs by the inhalation of condensed air.*

The benefit resulting from these proceedings is not of a merely temporary character, lasting only so long as the apparatus is in use; the changes are frequently permanent, especially if the treatment be persisted in for some time and the disease be not of a kind involving the destruction of tissue. Thus there are numerous instances on record in which pulmonary emphysema has been much diminished, in less aggravated cases actually removed, by expiration into rarefied air continued for weeks and months; not only has the dyspnœa been abolished and the vital capacity greatly augmented (sometimes nearly doubled), but the lower margin of the lungs has also been observed to rise gradually to its normal level in the thorax. Equally favourable are the results of this pneumatic treatment in chronic bronchial catarrh, and in bronchial asthma (uncomplicated, or associated with emphysema or catarrh). In the early stages of phthisis also the capacity of the lungs may be permanently increased by the inspiration of compressed air.

Pneumatometry.

To Waldenburg also belongs the credit of having been the first to apply the estimation of the force exerted in respiration,—an operation which, till his time, was regarded as of merely physiological interest (Valentin, Donders, Hutchinson, &c.),—to the diagnosis of diseases of the lungs. The manometer in most common use consists of a glass tube open at both ends, bent double, and supported on a wooden stand; each vertical limb is about 270 mm. high and is half-filled by a column of mercury, the top of which marks the point assumed as zero. One limb is turned horizontally at its upper end and is inserted into a caoutchouc tube; this is connected with a mask which fits closely on the face, absolutely excluding the air from the mouth and nose. On drawing breath through the caoutchouc tube the column of mercury rises in that limb of the manometer nearest to the patient and sinks in the other, movements which are, of course, reversed in expiration. The extent of the fluctuation is read-off on millimeter scales, of which there are two, one on each side of the instrument, placed on the glass tubes, and reaching above and below the zero points. Obviously the numbers so obtained must be doubled, as the quicksilver descends in the one limb exactly the same distance that it ascends in the other.

It has been abundantly proved by the researches of the above-mentioned observers that in healthy persons the *power* put forth in *expiration exceeds that of inspiration*, usually by 20—30 mm. on the scale just described. The following table gives more nearly the limits within which the pressure of forcible inspiration and expiration may vary in health :—

	Inspiration.	Expiration.
	Mm.	Mm.
In moderately strong men	70—100	80—130
In very powerfully-built men ...	120—160	150—220
In women	50— 80	60—110

These points on the scale are reached only when the movements of respiration are executed rapidly and with the utmost exertion of the respiratory muscles; the mercury is then maintained at its maximum height for scarcely a second, and falls again immediately. When inspiration and expiration are performed slowly the manometer indicates a much lower pressure, and the mercury remains longer at its highest point, oscillating slightly upwards and downwards. For practical purposes the former method recommends itself to the investigator, as we thereby ascertain the positive maximum of inspiratory and expiratory power the patient is capable of exerting.

As these pneumatometric signs have such a wide range even within the bounds of perfect health, it is important to know at what point they overstep these limits and acquire a pathological significance. Generally speaking it may be inferred that an *absolute* diminution of pressure exists in those cases in which it is decidedly below the physiological minimum; further, in largely-built individuals the physiological *mean* should be taken as the standard, any downward deviation from which is to be regarded as indicating disease; above all, however, the comparison of the inspiratory with the expiratory manometric pressure furnishes the best guide to a decision in this matter.

The diseases of the respiratory organs may be divided into two principal pneumatometric types: in the first the conditions of health are reversed, the positive expiratory pressure being less than the negative inspiratory force, while the latter may be somewhat increased, normal, or subnormal; in the second the negative inspiratory pressure is lower than the physiological minimum, while the positive expiratory force may be normal or subnormal, in the latter case being still slightly greater than the inspiratory force. To the first class, that in which *the expiratory pressure is diminished*, belong *pulmonary emphysema* (the lowering of pressure being here due to diminution in the elasticity of the lung-tissue), *bronchitis* and *nervous asthma*. In the second class, in which it is particularly the *inspiratory power* that is lessened, are found *phthisis pulmonum* (the manometric difference being observable here even when the disease is of but slight extent), *stenosis of the larynx and trachea*, and generally those diseases of the respiratory organs which offer increased resistance to the expansion of the lungs (pneumonia, pleurisy); in the more advanced stages of such cases the expiratory pressure may also be decreased. Among diseases of the heart mitral lesions most usually produce diminution of expiratory power, the result of the consecutive hyperæmia of the lungs (Waldenburg). Tumours of the abdominal organs (pregnancy, effusions, &c.), inasmuch as they impede expiration rather than inspiration, tend chiefly to lessen the expiratory force (Eichhorst).

It is worthy of notice that in comparing the pneumatometric with the spirometric signs, in health and disease, we find no evidence of any fixed relation between respiratory pressure and vital capacity; the former may be high while the latter is low, and *vice versa*.

PALPATION OF THE THORAX.

The phenomena discoverable by palpation, with the exception of those elicited in the examination of painful spots, do not belong exclusively to this method, most of them being recognisable also by means of auscultation and percussion. Palpation is therefore most commonly resorted to merely as a means of confirming or modifying the results of other methods of examination.

1. Movements of the Thorax.

The movements of the chest in inspiration may be as accurately ascertained by palpation as by inspection and mensuration. The palmar surfaces of the hands should be placed on the sides or on the anterior and posterior surfaces of the chest according as we wish to observe its lateral or antero-posterior movements; in this way even a very slight lagging-behind of one or other side is easily detected, frequently more readily than by inspection.

2. Examination of Painful Regions.

The seat of a painful sensation is often very vaguely indicated by the patient and can usually be determined with accuracy only by the application of the finger. Pain arising from *affections of the bones* forming the framework of the thorax, such as periostitis of the ribs, is aggravated by pressure. *Pleuritic* pain, which may quite escape the notice of the patient if the irritation of the pleura be not very intense, is frequently elicited only by pressure in one or more intercostal spaces and is always thereby considerably augmented; deep inspiration and forcible expiration, as in coughing, produce the same results. Pain in the intercostal nerves, *intercostal neuralgia,* is similarly increased by pressure. The pain of pleurisy may be distinguished from that of intercostal neuralgia by the fact that in the latter it generally extends along the whole course of the nerve in the intercostal space or becomes peculiarly acute on exercising pressure at certain spots ("painful points.") Pain in the muscles of the thorax, *muscular rheumatism,* is invariably aggravated by compressing the muscles between the fingers in a direction transverse to that of their fibres; occasionally it is felt only when this is done, and it is further never so perfectly localised as the pain of pleurisy or neuralgia. Patients sometimes complain of undefined uneasy sensations in the chest which it is impossible to trace clearly to any of the above-named causes; these are most probably propagated from some organ or nerve situated in the immediate neighbourhood.

3. Pectoral (or vocal) Fremitus.

By this term is designated a vibration of the walls of the thorax felt whilst *speaking*, singing, or screaming, by placing the hands on the chest. The vocal cords, thrown into vibration by the expiratory current of air, transmit these vibrations to the whole column of air in the bronchi and, if there be no special obstacle in the way, to the walls of the bronchi and through them to the thoracic parietes. The *intensity of the pectoral fremitus* depends, apart from the mere strength of the voice, on the following conditions: 1st, *On the pitch of the voice;* the vibrations of the vocal cords in the production of a low-pitched tone being necessarily *larger* than those concerned in the production of a tone of high pitch, the sonorous wave is carried outwards with greater force; just as in musical (stringed) instruments the vibrations of the lower strings are much more perceptible than those of the upper strings, being fewer in number in a given time, so in the human voice difference in tone gives rise to variety in the strength of the vocal fremitus, the latter being more marked in men than in women, most distinct in those having bass voices, and most feeble in high sopranos. 2ndly, *On the diameter of the bronchus into which the vocal vibrations are conducted, and its position relatively to the thoracic wall;* it is most intense therefore on the right side, the right principal bronchus being wider, joined to the trachea at a less acute angle, and situated nearer to the vertebral column than the left, which is also separated from the spine by the œsophagus and the aorta. 3rdly, *On the magnitude of the resistance offered to the passage of the sonorous vibrations through the walls of the chest;* the vocal thrill will therefore be most perceptible in patients in whom the subcutaneous fat is scanty and the muscular system but slightly developed. 4thly, *On the distance of the spot under examination from the larynx;* the fremitus is strongest over the larynx, is distinctly appreciable in the upper parts of the chest (in the clavicular regions in front, and between the shoulder-blades behind), and diminishes in force inferiorly.

Under certain *pathological* conditions the pectoral fremitus is *diminished* or even abolished, at other times it is *increased*, these modifications being rarely bilateral, usually confined to one side, and extending over a greater or less area of the chest-wall.

It is *diminished* by the effusion of a large quantity of fluid into one side of the chest, and *disappears* entirely when the exudation fills the cavity of the pleura and causes complete collapse of the lung. In the latter case the total suppression of the fremitus is obviously due to the fact that the vocal vibrations are but feebly conducted through the compressed bronchi and are still further weakened and diffused in the mass of the fluid effusion. The pleuritic exudation here checks the sonorous waves, acting exactly like the damper of stringed instruments, or the cloth covering which is placed on a tuning fork to deaden its sound.

Effusion which is small in amount, not encysted, limited to the lower and posterior part of the pleural sac, has little effect on the vocal fremitus; a decided diminution in its intensity takes place only when the thickness of the exudation reaches or exceeds 2½ cmtr. In cases of double pleurisy with effusion, in which the quantity of fluid thrown out is usually small and occupies the lower and hinder part of the chest, the pectoral fremitus is slightly feebler on both sides.

Diminution of the vocal thrill in the lower and posterior part of the thorax is of great value as enabling us to distinguish between pleurisy and pneumonia in the inferior lobe of the lung when the other symptoms alone are not sufficiently pronounced to warrant an exact diagnosis; it clearly indicates the presence of the former affection, as in the latter the fremitus is never decreased but always increased.

In the stage of absorption of pleuritic exudation the pectoral fremitus, which had previously been abolished, becomes again perceptible. This sign is of considerable importance, as it may sometimes be observed when the other symptoms of absorption are still absent; more especially the level of the fluid may remain unaltered though absorption has gone on to a considerable extent, the mass of exudation being simply reduced in thickness and the resistance to the propagation of the vocal vibrations in that way lessened.

Similarly in pneumothorax the vocal fremitus disappears because the sonorous vibrations penetrate the lung with difficulty and are arrested in the gaseous medium interposed between the lung and the chest wall. A slight diminution of the thrill is frequently noticed when the bronchi are loaded with secretion from the mucous membrane, as in chronic bronchial catarrh, the cause here also being that the abundant secretion presents an obstacle to the entrance of the waves of sound into the air-passages; after free expectoration the fremitus returns.

When the parenchyma of the lung is consolidated by infiltration and so rendered void of air the pectoral fremitus is *increased*, the condensed lung-tissue being an excellent conductor of sound. In normal conditions the voice is heard only very faintly over the side of the chest, as the healthy lung, consisting, as it does, of non-homogeneous tissues,—air-spaces and fibrous septa,—offers continual interruption to the outward passage of the waves of sound; when, however, the lung is infiltrated and impermeable to air it becomes a firm, homogeneous body, and acquires a higher conducting power. Further, the vibrations emanating from the vocal cords, when transmitted to the bronchi of a condensed portion of lung, are concentrated, being prevented from entering the infiltrated alveoli; they must thus reach the surface of the thorax in an exaggerated form. But it does not invariably happen that the pectoral fremitus is increased when the pulmonary tissue is consolidated; that this may be the case it is necessary that the bronchus leading to the part of the lung involved be in free communication with the trachea; should this communication be interrupted, as by superabundant bronchial secretion, the fremitus completely disappears, but returns when the mucus is expelled by coughing. Amongst those diseases which cause condensation of the lung-tissue, that in which the vocal fremitus is most frequently and most markedly augmented is the stage of *hepatization in pneumonia;* here the impermeability and solidification of the parenchyma are most pronounced, both as regards intensity and extent. Condensation from other causes is generally less complete; and in certain cases (of caseous pneumonia for example) it is not unusual to find that the voice, from concomitant disease of the larynx, is very feeble even over the vocal cords, so that its vibrations are no longer conveyed with distinctness to the surface of the chest.

detected by the ear than by the hand, the pleura becomes inflamed and covered by fibrinous deposit when the cheesy pneumonic process going on in the apex approaches sufficiently near to the surface of the lung. Pleural fremitus varies considerably in its duration; it sometimes remains only a few days at one part and then shifts to another according as the level of the fluid in the thorax alters, at other times it is fixed for a longer period at one spot; fremitus may thus persist for weeks or months according as the process of absorption is more or less prolonged. The differential diagnosis between pleural fremitus and other similar sensations connected with the respiratory apparatus is given below.

5. Bronchial Fremitus,

(vibrations caused by the movement of fluid secretion in the bronchi).

If the bronchial mucous membrane be extensively swollen, and the calibre of the bronchi in that way reduced, or if the air-passages be loaded with a very abundant fluid secretion, the entrance and exit of the current of air are obstructed; the bronchi are thus thrown into vibration and the fluid contained in them set in motion by the air as it passes through. The bronchial vibrations and the movements of the secretion are transmitted through the lung to the parietes of the chest, and these take the form of the fremitus under discussion, a very exact conception of the peculiar character of which may be obtained by applying the finger to a bass string which is vibrating powerfully.* As bronchial fremitus, when present, usually manifests itself over a large area, sometimes over the whole of the thorax—though possibly not with equal intensity at all points,—it is necessary, in order readily to ascertain its extent, to lay the palmar surfaces of both hands first on the back and front and then on the sides, directing the patient at the same time to make several deep inspirations. It may be of much the same intensity both with inspiration and with expiration, though occasionally it is more distinct with the latter,—a circumstance which is explained by the unusual slowness of expiration when the calibre of the bronchi is reduced by widespread catarrhal swelling of the mucous membrane.

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* I would recommend the term *bronchial fremitus* to designate the impression above described, especially as it presents an analogy with the expressions *pectoral fremitus* and *pleural fremitus*.

Bronchial fremitus is distinguished from pleural fremitus by the following points: it has none of the irregular, jerky character which marks the latter, in expiration it is as strong as, or even stronger than, in inspiration, and it may be temporarily diminished or even made to disappear by violent coughing, and especially by expectoration of mucus; in some other cases, in which the cough is unaccompanied by expectoration, the fremitus becomes weaker at certain parts of the chest but intensified at certain other parts,—a phenomenon which has its origin in the fact that forcible coughing displaces the fluid bronchial secretion and causes it to occupy less space than before. Pleural fremitus, on the other hand, is not at all modified by cough. (Various other diagnostic characters are revealed by auscultation, which see.)

Bronchial fremitus indicates the presence of *diffuse bronchial catarrh.* This agitation of the bronchial secretion is not only perceptible to the hand but is also audible, even at some distance from the patient, as a large bronchial râle. (See chapter on Râles.)

It is usually one of the most prominent symptoms in the bronchial catarrh of young children, as the mucus secreted in that disease, from the inability of patients of tender years to expel it by the act of coughing, accumulates largely in the bronchi.—The part at which the fluid secretion has chiefly collected may be determined with a certain degree of probability by simple palpation, the fremitus being at such points rougher and of a more rattling character; an exact diagnosis, however, is attainable only by means of auscultation. (See chapter on Râles.)

6. VIBRATIONS DEPENDENT ON THE MOVEMENTS OF FLUID SECRETION WITHIN PULMONARY CAVITIES.*

The agitation which is caused by the movements of respiration in the fluids contained in cavities in the lungs is communicated to the walls of the thorax, but only when these cavities are situated in the *upper* lobe, and near the surface of the lung, and when the chest itself is much *emaciated.* These vibrations differ markedly from those which have just been under consideration; they are feebler and finer, give the impression of the bursting of small bubbles, and are usually noticeable only at the end of inspiration, in the *upper part* of the chest in *front,* and over a limited area. They are entirely wanting when the excavation has taken place in the lower lobe, as the thickness of the muscular covering in that region offers great resistance to their transmission. Cough, especially when followed by expectoration, weakens them very much, or abolishes them for a time. (See chapter on Râles.)

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* [Cavernous Fremitus.—Trans.]

7. FLUCTUATION IN THE THORAX.

In cases of pleurisy in which the effusion is very considerable in quantity, almost completely filling one side of the chest, a feeling of fluctuation may sometimes be elicited by placing the palmar surface of one hand on the side or back of the thorax and tapping on the front with the finger of the other hand.

One might *a priori* expect to find fluctuation in such a case; occasionally, however, the rigidity of the thorax is such that the force imparted by the finger does not reach the fluid, or the chest-wall does not yield to the wave even when the fluid is set in motion. The presence of a thick covering of exudation on the pleuræ may also prevent the production of fluctuation,—a further consideration which also explains the comparative rarity of the phenomenon.

PERCUSSION OF THE THORAX.

HISTORICAL NOTE.

The discovery of thoracic percussion we owe to Auenbrugger, who was born at Gratz in 1722 and died at Vienna in 1809. When engaged in the study of empyema and the indications for the practice of thoracentesis he learned in 1753 to distinguish the healthy from the diseased side by the different sounds which they yielded to percussion. After having worked at this subject about seven years he published, in 1761, his "Inventum novum ex percussione thoracis humani ut signo abstrusos interni pectoris morbos detegendi." Auenbrugger himself recognised the value of his new method of exploration in the diagnosis of diseases of the chest,—though he had no conception of the wide application it was to receive in our day,—and insisted on its importance in his "Monitorium" addressed to his fellow-physicians. It was, nevertheless, much neglected by them: to some it remained quite unknown, by many it was confounded with the Hippocratic *succussion* observed in pyopneumothorax and accordingly ridiculed as the "Inventum novum antiquum," while by others (van Swieten and de Haen) it was set aside as unworthy of serious consideration. It was only by a very few, the principal of whom was Stoll, that it was employed in practice; and when the latter died in 1787 the great discovery passed into utter oblivion. Not until 1808, shortly before Auenbrugger's death, did the "Inventum novum" become generally known to German physicians, through Corvisart's French translation. Corvisart extended the application of percussion to the diagnosis of cardiac diseases and aneurism of the aorta. But it is to Piorry and Skoda that the most important advances in the study and practice of percussion are due. The former invented the Pleximeter (in 1826), and was also the first to avail himself of percussion in the examination of the abdominal organs; the latter clearly traced the special qualities of the percussion-sounds to their general physical causes, he originated the doctrine on which all our notions regarding percussion in normal and pathological conditions are based, and added to our knowledge of the sounds that may be produced by striking on the thorax by his exhaustive researches on the subject of tympanicity (1839).

For many details concerning the interpretation of the various qualities of the percussion sounds we are indebted to Wintrich (tympanicity), Traube (the pitch of the sound), Biermer, Geigel, Wintrich, Gerhardt (variations in the pitch of tympanitic and amphoric sounds), and many others.

Wintrich invented the percussion-hammer in 1841.

METHODS OF PERCUSSION.

There are two methods, *immediate* and *mediate*.

1. *Immediate* percussion, the method employed by Auenbrugger and also for some time by those who came after him, is performed by striking the thorax directly with the points of the fingers. In this way the more obvious of the differences in the intensity of the percussion sound are easily enough recognised. We thus obtain in the upper part of the chest a sound which we at once perceive to be distinctly clearer than that of the hepatic region; and we may also, by this means, define the various organs from each other with considerable exactness, and succeed in determining the presence and extent of many pathological conditions, such as pleuritic exudation, pneumonic hepatization, &c. The sound elicited by immediate percussion is loudest over the osseous portions of the thorax, more especially over the broad smooth surface presented by the sternum. Much less clear is the tone yielded by the direct percussion of the softer parts,—the intercostal spaces, the supra-clavicular and supraspinous regions, and the abdomen. This method has now, however, been generally abandoned, as it does not enable us to distinguish with sufficient precision between the finer shades of difference in the pitch or quality of percussion sounds.

2. *Mediate* percussion. It may be practised in three different ways,—with the finger of one hand interposed between the body and the percussing finger, with the finger and pleximeter, or with the hammer and pleximeter.

a. The method which consists in applying the second or index finger of the left hand to the skin and striking it with the middle finger of the right is best suited for the examination of the irregular or uneven parts of the thorax, to which the pleximeter cannot be made to adapt itself accurately; it is thus applicable in cases of depressed sternum, of undue prominence of the ribs, (particularly in emaciated persons), when the intercostal spaces are narrow, and we wish to define sharply the boundary line between solid organs and those permeable to air.

b. As the interposed finger is apt to become the seat of considerable pain and swelling if frequently percussed upon a pleximeter is usually substituted for it.

This instrument is commonly made of ivory, though sometimes vulcanite or other hard substance is used in its construction. The most useful form of pleximeter is an oblong, slightly oval plate of simple ivory, about 2½ cmtr. in breadth, and fitted at each end with small vertical ears roughened on their outer aspect. The accurate delimitation of organs is much better accomplished by narrow than by broad pleximeters. Instruments made with movable metallic handles should not be used, as the handles become loose and make a clattering metallic sound when the plate is struck. In using the pleximeter it is above all necessary to see that it is closely applied to the surface of the body, especially when examining uneven parts, as otherwise a small quantity of air may lodge between the instrument and the skin, the disturbance of which by percussion may give rise to accessory vibrations.

In percussing parts whose surface is very unequal the double pleximeter proposed by Seitz, made of caoutchouc and shaped somewhat like the bent tongue spatula, will be found very convenient. In such cases also we may percuss on the end of the finger, which forms a pleximeter having the advantages of being easily adaptable to all parts and of being small enough to serve for minute examination. The same object is accomplished by placing the pleximeter not on its flat side but erect, endways, and percussing on one of the fixed ear-like handles should these be large enough to answer the purpose; in such circumstances, however, percussion with the fingers alone is easier and gives the most trustworthy results.

In percussing, both on the finger and on the pleximeter, the finger with which the stroke is delivered should always be half-bent, while the movement of the hand should be entirely from the wrist. The proper execution of this manœuvre demands a degree of skill which is acquired by many only by long practice; those, however, who are accustomed to play on musical instruments, especially the piano, already possess the necessary command over the wrist.

c. Percussion performed on a pleximeter with Wintrich's hammer. This method is the easiest to learn, it is, as the finger is not used, the least disagreeable to practice, and by it louder and more definite sounds may be educed than when the finger is used as the striking agent. Very marked differences in resonance, such as that between the lung and liver sounds, are most clearly demonstrated in this way. Nevertheless, with this powerful percussion-sound is associated the great disadvantage that in it the finer degrees of difference in tone are completely lost. It may be laid down as a rule that the stronger the percussion the larger the area over which the vibrations are distributed. It is on account of possessing more of this penetrative power that hammer-percussion is apt to lead to erroneous conclusions, that a clear sound may be produced even though the medium immediately behind the part struck be void of air, the neighbouring air-containing structures being thrown into vibration; this source of error has to be specially guarded against in examining the cardiac and splenic regions. Another objection to the practice of hammer-percussion is that it involves the loss, to a great extent, of the sense of resistance which is experienced in finger-percussion. Both disadvantages may be partially overcome by percussing with a light stroke, and by grasping the hammer, not at the end, but at the middle of the handle, and at the same time keeping the index finger closely applied to the hammer-head; the finger is thus brought nearer to the percussed structure, and the sense of resistance communicated by the latter more readily detected. When the utmost precision is demanded in mapping out the size of organs and in tracing the boundaries which separate parts containing air from those which do not, when, for instance, we propose to define with rigorous exactness lung from liver, heart, spleen, &c., it is better to use the finger rather than the hammer, as by this *tactile* system of percussion the slight resistance of parts permeable to air and the greater resistance of those which are impermeable, are most distinctly appreciable.

Whatever be the method of percussion adopted, if the examiner have acquired sufficient skill in its performance an absolutely accurate result may always be obtained.—Although hammer-percussion, being the most easily mastered, is most generally employed, it will be found advantageous to percuss always with the finger and pleximeter so long as we can thereby elicit a distinctly defined sound. *He who is skilled in digital percussion will be able to percuss equally well with the hammer,—an axiom the inverse of which does not hold good.* It is also obvious that besides being proficient in the technical part of the proceeding it is necessary to possess a sensitive ear, educated to distinguish between the finer gradations of sound.

Rules regarding the employment of *forcible* or *gentle* percussion can be set forth only in a general way.—The stroke should be somewhat *forcible* in examining those parts of the chest which are covered by a thick layer of fat or muscle,—the mammary and supraspinous regions,—in order that the vibrations may be carried through them to the lung beneath; gentle percussion in these parts would not give the true pulmonary resonance but only the dull sound of the superficial soft parts. Similarly, a *strong* percussion-stroke is necessary to determine the condition (as regards the *presence of air*) of *deeply-seated structures*, lying below other internal organs or tissues which may or may not contain air. (See p. 79).

On the other hand percussion must be *gentle* where *solid* and *air-containing organs* (lung and liver, lung and heart) *border on each other* superficially, and in all cases in which it is desired to ascertain the density of parts situated immediately below the surface. The line of demarcation between lung and liver may thus be most sharply defined by percussing downwards with so little force that on reaching the liver only the sound caused by striking the pleximeter is heard; just at this point, however, at the end of a full inspiration the lower border of the lung encroaches slightly on the liver, and we then again obtain the clear pulmonary sound. In this way we find that the ear appreciates more promptly the difference between *absolute dulness* and *slight resonance* than that between the *greater* or *less clearness* of the tone produced by more forcible percussion.—The percussion-stroke must also be less energetic in children than in adults, because in them the thorax is more yielding and the internal organs are smaller, so that the vibrations generated by percussion at any part are more widely conducted through the tissues, and in that way the more delicate alterations in the nature and quality of the sound are hidden; thus it often happens that in percussing the chest in children a powerful stroke is answered by a sound of a tympanitic character proceeding from the simultaneous vibration of air in the intestines.—It is advisable also to percuss less vigorously in the vicinity of inflamed and painful parts, over pulsating aneurisms (to avoid the danger of bursting them) and cavities in the lungs; rough treatment of patients in whose lungs destructive processes are going on gives rise not only to pain but frequently also to attacks of coughing. Those who have shortly before suffered from hæmoptysis, or are still bringing up blood in the expectoration, should not be subjected to examination by percussion; cases —usually phthisical—are not wanting in which hæmoptysis has followed repeated percussion of the chest performed for the purpose of practical clinical teaching.

One of the most important rules is always to percuss the thorax *symmetrically* on the two sides. Although the more marked deviations from the normal resonance are usually at once appreciated by the ear, it is only by careful comparison with the sounds given by the corresponding part on the healthy side that the slighter variations can be satisfactorily made out. Thus in examining the lungs in front they should be percussed symmetrically from the supraclavicular regions downwards to the fourth rib, at which point the heart is encountered on the left side; laterally and posteriorly the same comparative method of examination may be continued downwards quite to the lower borders of the lungs. The muscles on both sides should as nearly as possible be in the same state of tension; if they be more tense or prominent on one side than on the other the resonance will be somewhat diminished on that side. In percussing the back of the chest the arms should be crossed in front in order to increase the space between the shoulder-blades and to ensure that the muscles of the back shall be in an exactly similar state of contraction on both sides. In like manner in percussing the supraclavicular fossæ the patient must hold his head erect, with the face looking directly forwards; should he incline it to one side, as he is apt to do for the convenience of the examiner, the sound becomes less clear from the increased tension of the soft parts of the neck. So far as the percussion of the lungs is concerned it is immaterial whether the patient assumes the sitting, standing, or recumbent posture.

THE THORACIC PERCUSSION-SOUND.

This sound is of a complex nature. *It is produced essentially by the vibration of air contained in the pulmonary air-vesicles, in part also by the vibration of the thoracic parietes,* and is to a certain extent influenced by the degree of tension of the parenchyma of the lungs. That the normal clear percussion-note depends principally on the vibration of air in the lung is proved by the facts that it becomes decidedly duller as the capacity of the lung diminishes, and that when a lung which has become quite impermeable to air (*e.g.*, consolidated by pneumonic hepatization) is extracted from the thoracic cavity and percussed it gives a perfectly dull, almost inaudible, sound. That the vibration of the chest-wall also is concerned in the production of the sound under discussion is indicated by the circumstance that the lung, when removed from the chest and inflated, is less resonant than when still within the thorax. And lastly, that the percussion-sound is to a certain extent modified by the varying state of the lung as regards the tension of its tissue, is shown by some pathological observations, to be mentioned further on.

Many authors deny that the chest-wall participates in any degree in the causation of the percussion-note, by others the part it plays is much over-estimated, while by some it is considered the only part concerned in the production of the sound. Williams explains the normal thoracic resonance *exclusively* in this way; he believes that the vibrations of the thoracic parietes, which are readily propagated through a lung filled with air, are disturbed in their transmission or completely arrested by an impermeable lung, or by the presence of fluid or solid exudation in the pleural sac,—just as the tone emitted by a violin-string becomes feeble when the mute is placed on the bridge of the instrument. Such a theory is fitted to explain only the diminution in the intensity of the sound in condensation of the lung, though it is not to be forgotten also that a hepatised lung, when taken from the chest, is just as non-resonant as before its removal; it does not, however, satisfactorily account for the production of tones having a tympanitic and metallic timbre. Williams' theory requires no detailed refutation. It is evident, on the other hand, from the following considerations, that the chest-wall *does contribute* in some degree to the formation of the percussion-sound: Mazonn has shown that when the free vibration of any part is prevented by the pressure of the hand or of weights laid on the surface the note obtained in the region concerned loses in resonance, even after extraction of the thoracic organs; increase in the tension of the ribs or skin raises the pitch of the sound, decrease in the tension of the ribs (as after removal of the sternum) lowers it; the elevation in pitch which accompanies deep inspiration depends chiefly on the greater tension of the ribs caused by the expansion of the chest and on the contracted state of the muscles; this raising of the pitch also occurs at various parts behind which no portion of the lung is situated,—a fact which negatives the idea that the inspiratory expansion of the lungs has anything to do with it; it is moreover well known that increase in the volume of the lungs in inspiration has the opposite effect,—it lowers rather than raises the pitch (Rosenbach; see p. 88).

THE PROPERTIES OF THE PERCUSSION-SOUND.

The classification of the properties of the percussion-sound is based on that of the qualities of a musical tone, though strictly speaking these two species of sounds cannot be regarded as absolutely identical with each other in all respects. A musical tone is described as possessing Pitch, Timbre, and Intensity.

The *pitch* of a musical tone varies with the number of vibrations (as of a string or a column of air) which take place in a given unit of time.—Its *timbre* depends on the construction of the musical instrument, all those made of like materials (stringed instruments, for example) giving tones of an exactly similar character; timbre is also slightly different according to the particular variety of instrument used, so that a cultivated musical ear is able at once to tell whether a given note has been sounded on a violin or violoncello.—The *intensity* of a musical tone depends on the amount of force expended in producing the sonorous vibrations, and consequently on the amplitude of the latter.

The three qualities which distinguish musical tones are not found in the *normal* thoracic percussion-sound,—it possesses pitch and intensity, but not timbre; it is not, in fact, a musical tone in the sense in which that expression is used in physics, but is better described simply as a *sound*. As this normal sound, however, does acquire timbre, the tone ("klang") which renders it of musical quality, in certain pathological conditions, a third property must be ascribed to it,—that of *absence of timbre* (want of distinct musical character, non-tympanicity). Of these three qualities,—intensity, pitch, and absence or presence of the peculiarity called timbre,—the most important is the first.

THE INTENSITY OF THE PERCUSSION-SOUND.

(Clearness and dulness.)

The opposite extremes in the intensity of a sound are strong and weak, loud and soft; but in the terminology of percussion which prevails in Germany, the extremes are designated *loud* and *dull*, the intermediate varieties of resonance *muffled* or *obscured*.

However convenient this nomenclature may be, if we wish to indicate the qualities of the percussion-sound as far as possible in terms similar to those employed in describing musical tones the above expressions must be considered badly chosen, as *dull* is not the opposite of *loud* but of *clear*. On the other hand *clearness* and *dulness* are not the opposite extremes of *intensity* of a tone, because a clear tone may be very weak (soft) and a dull one very loud; nevertheless *loud* and *dull* may be used to designate contrary qualities of a tone with respect to timbre and pitch.—It would thus be more appropriate to speak of the normal pulmonary sound as being both *loud* (strong) and *clear*, the opposite of which would be *soft* (weak) and *dull*; in this way we should indicate both the intensity and timbre of the sound. The term soft ("*leise*"), however, is one that has never been definitely adopted into the terminology in question.—*Muffled* is a very fitting term by which to express the transition from loudness (clearness) to dulness, as a muffled sound is not only less loud but also less clear.

Apart from the mere force exerted in percussion, the intensity of the sound depends, 1st, on *the structure and thickness of the chest-wall;* and, 2ndly, on *the quantity of air contained in the lungs*.

1st. The shock produced by the percussion-stroke is much diminished in force in its passage through the thoracic parietes to the lungs, this enfeeblement during transmission being the more marked the thicker the tissues through which the impulse has to be conveyed; frequently, therefore, the amount of vibration set up in the lung is but very slight. Thus the sound that is given by the lung, even when the latter is in a normal condition as regards air-contents, is always somewhat duller at those parts at which the subcutaneous fat is abundantly developed; in the mammary region in females, when the breasts are large, it is almost absolutely dull, especially on percussing gently; it is also less clear over those parts occupied by large masses of muscle, as over the whole posterior surface of the thorax, especially in the supraspinous regions; it further diminishes in intensity when the covering of the thorax is thickened by œdematous swelling. Similarly, the percussion-sound becomes duller, sometimes even to a considerable degree, when the ribs are strongly curved and the chest-wall thereby increased in thickness; this is especially noticeable on the posterior surface of the thorax in those suffering from kyphosis.

2. The sound is *muffled* when the quantity of air in the chest suffers diminution, and is rendered positively *dull* when the substance of the lung becomes completely *impermeable to air*. The sound obtained on percussing a lung which is entirely void of air is quite indistinguishable from that yielded by any other solid organ, such as the liver or the *thigh;* the typically dull sound is therefore sometimes called the *femoral* sound.

The decrease in the volume of air in the lung, however, must be considerable, and must implicate the organ somewhat extensively before it can produce any very sensible effect on the percussion-sound; a very slight diminution does not alter it in any degree. In percussing a lung removed from the body and powerfully inflated we obtain a clear sound; after allowing a small quantity of the air to escape we find that the sound remains quite as clear as before. In like manner it is frequently observed that in the beginning of acute, or in the course of chronic, diseases of the lungs the percussion note remains unaffected, notwithstanding the marked falling-off in the volume of air which the lung is capable of accommodating, a diminution which is the inevitable consequence of the nature of the morbid process going on, and the existence of which is proved also by certain auscultatory signs to be subsequently considered.

It is necessary, in order to the production of any marked decrease in the intensity of the percussion-sound, that the portion of lung rendered less permeable to air should have an area of at least 4 square cmtr., and that it should lie near the surface. *Very circumscribed* portions, even when situated superficially and perfectly consolidated, do not modify the sound, and still less is this effect produced by more deeply-seated lesions, even when they are of considerable extent. In the first case the sound continues unchanged because it is impossible to confine the vibration caused by the percussion-stroke to an area so limited, in the second case because the condensed parenchyma is at all points covered by lung tissue which contains air and which gives a clear sound.

It has already been stated (p. 74) that in order to detect deep-seated consolidation it is necessary to percuss with a fair amount of force, when it is found that the tone rendered by the part in which such consolidation occurs is less loud than usual; positive evidence of its existence, however, is attainable only when it is comparatively near the surface and involves a somewhat large portion of the lung. The generally-accepted view, that this muffling of the resonance over those parts of the lung in which consolidated tissue is sheltered behind tissue that is freely permeable is due to the diminished depth of the stratum of air thrown into vibration, is supported by the fact that the percussion-sound of large portions of lung removed from the body and fully distended is clear, that of smaller pieces being somewhat duller. This is in accord with Weil's statement that the dulness is not owing to any quality communicated to the sound by the solid part, —a theory that is founded on the following simple experiment; Weil found that two portions of lung of equal size, removed from the body, gave an equally clear sound to percussion, and that the relation was not disturbed even when one of the pieces was placed on a solid substance, such as the liver.

When the ear is accustomed to the normal pulmonary percussion-sound very marked dulness is at once recognised without any comparative examination of the healthy side, though it is only by the latter method that the slighter deviations from the normal resonance are detected. Should the sound be of nearly equal intensity at two symmetrical points,—above or below the clavicles for instance,—percussion of the surrounding parts must be trusted to show whether or not it is normal.—Obviously the stroke must be equal in force on the two sides.

The diseases of the respiratory organs associated with *decrease in the intensity* of the percussion-sound may be arranged in two groups, those in which the air vesicles are *infiltrated* with plastic exudation, and those in which they are *subjected to pressure*, and thus temporarily or permanently closed (by the presence of fluid or tumours in the pleuræ).

1. *Dulness* due to *infiltration of the lungs*.

In pneumonia in the stage of hepatization the percussion-sound becomes dull, the alveoli of the lung being completely filled with a fibrinous exudation which displaces the air they normally contain. The more nearly this hepatization, which usually involves one entire lobe,—most commonly the lower,—approaches the surface of the lung the more marked is the dulness, so that frequently the latter closely resembles the perfectly dull liver-sound. The absence of resonance, on the other hand, is less appreciable when the consolidated parts are separated by healthy lung tissue, and the sound suffers no sensible diminution if the induration be in very small isolated patches (see p. 79), even when these are superficial; and similarly when the affection, though comparatively extensive, occupies the central parts of the lung, as in central pneumonia, the percussion-note remains quite unaltered.

In the first stage of pneumonia, when, though the pulmonary capillaries are engorged with blood, the alveoli are still free of exudation, the thoracic resonance is normal; it is only towards the end of this stage, when a certain amount of exudation has been poured into the air-cells, but not enough to completely dislodge the air, that the intensity of the percussion-sound diminishes; it becomes slightly muffled, and acquires a tone of a somewhat tympanitic character (see p. 99), from the relaxation of the pulmonary parenchyma; in the third stage the exudation disappears, the air again gains entrance into the alveoli, and we have the same physical conditions as at the end of the first stage—the presence of both air and fluid in the air-cells producing a dull percussion-sound, which gradually becomes clearer, and at the same time assumes a tympanitic quality as the absorption of the infiltration goes on. This tympanitic character is lost when the process of absorption is ended and the lung has returned to its normal state. In those cases in which the infiltration, instead of being absorbed, passes into cheesy degeneration, or a state of chronic induration, the capacity of the alveoli is permanently lessened, and the thoracic resonance is more or less diminished.—It is not unusual to find all three stages represented simultaneously in the chest of one patient, one part of the lung being hepatized, another part having reached the stage of resolution, and a third portion of the same or another lobe just taking on inflammatory action; it is in that way that we are able to explain the sudden transition from an absolutely dull to a muffled or even a clear sound in the immediate vicinity of consolidated portions of the lung.

Pneumonia occurs most frequently in the lower lobes of the lung, especially on the right side, more seldom in the upper lobes or right middle lobe, or in both lower lobes at the same time. If only a part of a lobe be consolidated the form of the non-resonant region, mapped out on the surface of the chest, presents nothing characteristic; in hepatization of an entire lobe, on the contrary, we are able to trace externally its exact outline. When the whole of the lower lobe is affected resonance is diminished posteriorly as far upwards as the middle of the shoulder-blades; in condensation of the whole of the upper lobe the dulness extends downwards to the fourth or fifth rib in front, to the fourth rib laterally, and is recognisable also in the supraspinous regions; in hepatization of the right middle lobe dulness is most readily detected in the axillary line, between the fourth and fifth ribs. —As a pulmonary lobe, when completely consolidated, increases considerably in bulk the region which it renders dull to percussion is correspondingly enlarged.

In certain cases pneumonia does not pass regularly through its three stages, nor does it confine itself to the locality in which it first appears; it *wanders* from one lobe or part of the lung to another, and resolution follows at once on the onset of the inflammation, without the occurrence of hepatization; the disease may thus run a course lasting several weeks, attacking successively all the lobes of the lung, and sometimes occurring in the same parts more than once.

In the pneumonia of children, which is seldom fibrinous, but usually catarrhal and following on capillary bronchitis, the thoracic resonance is never so strikingly diminished as in that of adults, because in the former the substance of the lung is not completely emptied of air, but contains islands of permeable tissue in the midst of the consolidated parts.

As in pneumonic hepatization, which is the type of complete consolidation of the lung, so in *every other variety of pulmonary condensation, from whatever cause arising*, the percussion-sound is *muffled* or *absolutely dull*. To this category belong the different forms of *cheesy pneumonic condensation of the lung* and *chronic interstitial pneumonia* usually designated by the collective term *phthisis*. The intensity of the dulness is determined by the degree of infiltration and blocking-up of the air-cells and by the extent of lung concerned; thus the thoracic resonance may be perfectly normal during the first stages of phthisis, and it certainly suffers but slight modification when patches of healthy lung tissue are still found between the condensed portions.—The apices of the lungs are the parts most subject to this caseous pneumonic degeneration; they may both be attacked, but seldom simultaneously, or with equal violence; at other times the disease is unilateral, occurring then most frequently on the right side. Dulness so produced is accordingly most commonly found in the supraclavicular regions and on the clavicles; if the infiltration have invaded the whole thickness of the lung, which is very frequently the case, the tone in the supraspinous regions also loses in resonance, and should it be more abundant posteriorly than anteriorly the dulness will be less marked in front than behind, or may even be absolutely wanting over the clavicles. When the condensation spreads gradually to the lower part of the superior lobe the dulness to percussion extends similarly to the infraclavicular region as far downwards as the third and fourth ribs, and also, though not to so marked a degree, to the upper part of the shoulder-blades.

Acute miliary tuberculosis never diminishes the resonance of the chest, as it does not cause consolidation of the lung substance.

Effusion of serum into the pulmonary vesicles (*œdema of the lungs*) gives rise to but very slight muffling of the percussion-sound, as the air-cells are not completely filled with fluid but still contain a certain quantity of air, the lungs being thus in a condition analogous to that presented at the end of the first stage of pneumonia.—Effusion of blood into the pulmonary vesicles (hæmorrhagic infarction) produces dulness to percussion only when the infiltration is so abundant as to totally exclude the air, and when the affected part is more than 4 cmtr. in diameter and situated near the surface of the lung.

Hæmorrhagic infarction, as the result of mitral lesion, occurs most often in the middle and lower lobes of the right lung. Infarction is also rarer in the centre than at the periphery of the lung.

2. *Dulness* due to *compression of the lung.*

This results generally from the presence of fluid in the pleural sac. A small quantity does not obscure the sound, this taking place only when the effusion has a thickness of at least 1½ cmtr. Experiments performed on the dead body, in adult males, showed that it is only after the injection of 400 ccm. of water into the pleura that the area of dulness rises two fingers'-breadths above the posterior lower margin of the lüng; when augmentation of the mass of exudation is attended also by increase of the diameter of the layer of fluid interposed between the lung and the chest-wall the percussion-sound becomes less and less clear, and when the layer measures more than 5 cmtr. in thickness the sound becomes absolutely dull. So long as the collapse of the lung from pressure is not complete we can, by percussing forcibly, demonstrate the presence of the residuum of air, the sound so elicited being distinctly more resonant.

Slight exudations gravitate towards the lower and posterior parts of the pleural cavities, so that it is to these regions that the alteration in the thoracic resonance is at first limited; as the effusion increases in volume it spreads to the sides, and then to the front, of the chest, when the non-resonant area is found to extend from the vertebral column behind to the middle line in front. The level of the fluid, as traced by percussion, is not perfectly horizontal, but is often higher posteriorly than at the side or in front. Further, as the layer of fluid is of less thickness in its upper than in its lower part the dulness is more decided towards the bases of the lungs. And finally, when the diaphragm is forced downwards, as it is, both laterally and anteriorly, when the effusion is very abundant, the non-resonance to percussion is found to be co-extensive with the displacement; thus, in exudation into the left pleura dulness is detected at the anterior and lower part of the thorax, where normally the tympanitic sound of the stomach is heard, while in exudation into the right pleura the dulness in the hepatic region is considerably increased. When the visceral and costal pleuræ have contracted adhesions in the course of a former attack of pleurisy, the fluid does not move freely in the pleural cavities, but is enclosed in such spaces as are left between the adhesions, forming the so-called encysted pleuritic effusion; in these circumstances the dulness has no characteristic shape, and a definite diagnosis is possible only by calling to our aid the other methods of exploration.

When the exudation is undergoing absorption the lung begins to expand again gradually, and the dulness diminishes—both in extent, as the level of the fluid sinks, and in intensity, as the thickness of the layer of effusion, and with it the distance between the lung and chest-wall, decrease,—the air which enters the lungs making its influence felt in clearing the percussion-sound. If, notwithstanding the complete absorption of the fluid or its discharge outwardly through perforation of the thoracic parietes or by operative puncture, the lung shows no tendency to return to its former dimensions, having become collapsed from persistent compression, the percussion-sound remains permanently dull.

Certain recent experimental investigations by Garland and by Ferber, consisting essentially of the injection of slowly-coagulating fluids into the pleural cavities of dogs, give results more or less confirmatory of the foregoing statements. A slight effusion first of all separates the peripheral part of the diaphragm from the thoracic wall, making room for itself in the complementary pleural sinus which, in ordinary superficial respiration, is not occupied by the lung; dulness begins to be appreciable only when the level of the fluid rises above the margin of the lung. The upper surface of the fluid has a constant tendency to run into the horizontal position, and alters therefore with each change in the posture of the body; its upper boundary is never perfectly horizontal, however, but presents many curves, while the mass of the fluid is continually rising and falling,—a fluctuating motion which depends to a certain extent on movements proper to the fluid itself, but principally on the movements of the respiratory organs and of the heart. It is possibly owing to these undulatory movements that when at a later stage peripheral adhesion occurs at the upper margin of the exudation it does not take place in a horizontal, but in a somewhat irregular or wavy, line. The position and outline of the mass of fluid, however, depend chiefly on the habitual posture of the body; thus it usually reaches to a considerably higher point behind than it does in front when the patient has been long confined to bed lying on his back, and it remains at this level on the formation of inflammatory adhesions, while its upper boundary is generally nearly horizontal when the patient is able to be up and to move about during the time that the exudation of lymph is going on.—Further, the distribution of the fluid, and the consequent conformation of the non-resonant area, are influenced to an important degree by the occurrence of partial adhesions between the visceral and costal pleuræ at an early stage in the disease, and also to some extent by the retractibility of the lung, this not being equal at all points even in health. In these considerations may be found the explanation of the fact that while most commonly the upper margin of the dulness is higher behind than in front in some cases it does not deviate much from the horizontal line.

What has just been stated with regard to pleuritic exudation is applicable also, with certain modifications, to the transudation of fluid into the pleural cavity, *Hydrothorax;* but whilst the former, with very rare exceptions, is unilateral, the latter is invariably double, not originating simply in inflammation of the pleuræ but in disease of the heart or kidneys. Cardiac diseases, mitral disorder for example, give rise to this transudation by producing an overloaded condition of the pleural veins, and renal disease by causing hydræmia. If the quantity of fluid transuded be not excessive the intensity of the percussion-sound may be considerably modified by changing the position of the body, as the liquid always sinks to the lowest part of the thorax. Thus in patients suffering from Hydrothorax the fluid stands at the same level in front and behind so long as they maintain the upright position; but when they lie on the back the fluid gravitates to the posterior parts of the chest, and the percussion-sound becomes clearer in front. (This change in the thoracic resonance from alteration of the position of the body is also frequently observed, but to a less marked extent, in cases of ordinary pleuritic effusion.) In general the transuded fluid, which is not usually very abundant, occupies only the posterior and lower part of the pleura and is scarcely observable in front; the dulness caused by it therefore does not equal in extent or intensity that of pleuritic exudation.

Solid bodies situated between the lung and the chest-wall have the same muffling effect on the percussion note. Amongst these must be reckoned tumours of the mediastinum. The sound is similarly rendered dull when the lung is subjected to pressure by the development within itself of certain morbid growths (carcinoma, sarcoma), by tumours of the bronchial glands, by marked hypertrophy of the heart, and by large tumours of the liver or spleen which push the diaphragm upwards. In these cases and in others of a like nature the non-resonant region, even when the dulness is of great intensity and distributed over a considerable surface, has not the outline which is so characteristic of pleural exudation, but is usually somewhat irregular in conformation; obviously also the dulness is to be attributed to the solid body which gives rise to the pressure rather than to the impermeability of the portion of lung so acted upon.

Diseases of the bronchi, unaccompanied by structural change in the substance of the lung, *never diminish the thoracic resonance;* the calibre of the bronchi may be very much reduced by swelling of their mucous membrane, by very abundant catarrhal secretion, or, as in croupous bronchitis, by firm fibrinous exudation, yet there is always sufficient room left to permit of the access of air to the alveoli; it is only when bronchial diseases invade the parenchyma of the lungs, exciting secondary infiltration and consolidation (broncho-pneumonia, bronchiectasis), that muffling of the percussion-sound is observed.

INCREASE IN THE INTENSITY OF THE PERCUSSION-SOUND.

Conditions the opposite of those which obscure the percussion-sound (see p. 78 *et seq.*) increase its intensity; it is clearer therefore when the thoracic parietes are thin (in patients, for example, whose muscles and subcutaneous fat are slightly developed), as the force of the percussion stroke is more readily and with less loss of intensity conducted through them to the lungs, in which organs, accordingly, more vigorous vibration is set up. In old people therefore, and in those who are much emaciated, the percussion note is loud and clear. If the loss of tissue be confined to one side (progressive muscular atrophy) the chest, as in a case which I had an opportunity of examining, in which the right pectoralis major had almost entirely disappeared, may be so resonant at that part that the normal sound given at the corresponding point on the healthy side seems almost dull in comparison. Full inspiration intensifies the percussion note, as the volume of lung thrown into vibration is larger; in quiet respiration such differences in the intensity of the sound are not appreciable. In pulmonary emphysema the chest is sometimes abnormally resonant (the tone being also usually somewhat higher in pitch), especially in severe cases. Biermer designates this the *Bandbox sound* [Schachtelschall]. There are no other conditions to be noticed in which the thoracic resonance is simply increased in intensity. No special diagnostic value, therefore, attaches to this sign.

PITCH OF THE PERCUSSION-SOUND.

The pitch of the percussion-sound depends on the degree of tension of the chest-wall on the one hand, and on that of the tissues of the lung on the other. The tension of both parts is increased in inspiration and diminished in expiration, so that in full inspiration the tone becomes slightly higher and in forced expiration slightly lower; and this same rise and fall in pitch is observed both in healthy and in diseased lungs. As has been already stated, the most important of the circumstances which determine the pitch of the percussion-note is the tension of the chest-wall, the least important that of the lung substance; that the latter indeed exercises but little influence in this respect is evident from the mere fact that the inspiratory rise in pitch above referred to takes place also in certain pathological conditions in which changes in the tension of the pulmonary parenchyma are scarcely possible, such as diseases involving destruction of tissue. —The simple increase of *volume* that the lungs undergo in inspiration, apart from the forementioned greater tension, takes no part in causing the simultaneous rise in the pitch of the percussion-note; it might rather, on the contrary, be expected that the sound should become lower during inspiration, as the volume of air in vibration is greater than during expiration. This lowering of the tone, however, does not occur, as any tendency in that direction is more than compensated for by the other factors,—by the increased tension.—When the breathing is quiet the pitch remains practically the same during both phases of respiration.

In the normal thorax differences in the pitch of the percussion-sound at different parts may be recognised by a cultivated musical ear; thus, on the right it is usually found to be deeper than on the left, though occasionally the reverse condition is met with. Even on the same side of the chest the sound is not of the same pitch at all points: in the neighbourhood of the liver it is somewhat higher than at other parts,—at least in the sitting and standing positions. A slight elevation in the pitch of the tone is noticeable on the front of the thorax on raising the body from the recumbent to the sitting posture, due, evidently, to the increased tension of the thoracic parietes; this phenomenon is observed also in the dead body, a fact which excludes Rosenbach's theory, that it is caused by change in the respiration.—The variations in the pitch of the percussion-sound in the thorax are occasionally so trifling as to be appreciable only when carefully watched for; in other cases they are so marked that inexperienced examiners not unfrequently mistake them for differences in intensity, regarding the grave sound as dull in comparison with the acuter or clearer sound yielded at other points. These variations possess no diagnostic significance, as they depend only on *physiological* differences in the tension of the chest-wall.

The percussion-note becomes *abnormally deep* in those pathological conditions which are accompanied by *diminution in the tension of the lung tissue* at any part: these are pleuritic exudation and pneumonia (Traube), of moderate intensity.

1. In *moderate pleuritic exudations*, occupying, for instance, not more than the half of the pleural cavity in front, laterally, and behind, the percussion-sound in the *infraclavicular* region * becomes deeper in pitch than at the corresponding point on the opposite side; this is caused by the gradual *retraction* of those parts of the lung still permeable to air and situated above the level of the fluid, by their slow return to their natural volume in the undistended state,—that is, by the loss of tension in those parts.

A retracted lung contains less air than one in its normal state, but the gravity of the percussion-note is not due to this, as diminution in the volume of air present in the lung usually raises the pitch of the sound, as is seen in almost every case in which the latter is muffled or dull.

2. In *pneumonia* also the pitch of the sound is lowered over those parts of the lung which remain *accessible to air*, and are situated near the hepatised portion. If, for example, the lung in its posterior aspect is completely consolidated while air is still freely admitted to the parts in front, the infraclavicular region gives an abnormally deep note on percussion. Here again, as in cases of pleuritic exudation, the same cause is in operation, deepening the sound,—decrease of tension in the non-infiltrated portions of the lung.

This lowering of the pitch of the percussion-sound is not invariably present in cases such as those described, as the retraction of the permeable parts of the lung does not always reach the precise degree which is favourable to the production of the phenomenon. Its duration must obviously be short in pneumonia, and it disappears rapidly in pleurisy also when the exudation undergoes any very great increase; I have observed it lasting only a few days, or even a still shorter time, in some cases in which the exudation was stationary. And further, at the commencement of the stage of absorption of very large effusions, when the compressed lung begins again to expand, the sound in the infraclavicular region may become abnormally deep.

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* When the *anterior* part of the upper lobe of the lung is entirely emptied of air, and the *posterior* part slightly reduced in volume (as in cases of encysted exudation, glandular tumours, &c., situated in front) the percussion-sound in the supra-spinous region is abnormally low in pitch.

The percussion-sound in the above-mentioned circumstances, in pleuritic exudation and in pneumonia, may be simply lowered in pitch, or it may also be attended by a tone of somewhat tympanitic character, or may even eventually become perfectly tympanitic; in another class of cases of the same diseases the note is tympanitic from the very outset, without having been previously lowered in pitch (see p. 97 *et seq.*).

Elevation of the pitch from *pathological* causes never occurs alone, unaccompanied by decrease in the intensity: the sound generally rises in pitch as it loses in clearness. An abnormally high-pitched percussion-sound is thus of no special diagnostic importance, as it never exists alone as a pathological phenomenon.

THE TYMPANITIC PERCUSSION-SOUND.

This sound owes its name to its resemblance to the sound of a drum, though the timbre in the two cases is not exactly identical. In its physical characters it closely approaches the musical tone,* inasmuch as it is a sound the pitch of which can be easily and accurately determined. The percussion-sounds of the larynx (with the mouth open or closed), of the distended cheeks, and of the stomach and bowel, illustrate well the differences in the pitch of the tympanitic note.

Tympanicity is not an unusual symptom in many of the diseases of the organs of respiration, but is never found on percussing the normal chest. In tracing the manner in which it is produced we must go back to its physical cause, which is most simply demonstrated by percussing the larynx or intestine, or by striking on the upper part of a drinking-glass, and so throwing into vibration the column of air contained in it.

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* Gerhard has tried to prove, by means of König's sensitive gas-flame, that the tympanitic tone is composed of a succession of similar and regular sonorous waves, the non-tympanitic sound, on the other hand, of dissimilar waves. If a tympanitic sound or a tone of low pitch be received in the funnel of the apparatus and directed on the flame, a series of equal indentations, with non-tympanitic sounds a series of unequal indentations, appears in the curved bands of light on the rotating reflecting prism. H. Jacobson, on the contrary, whose experiments I have had the pleasure of witnessing, denies that the tympanitic sound is distinguished from the non-tympanitic by any marked difference in the flame-tracing.

If we percuss with the hammer on a pleximeter held over the mouth of an empty or only partially filled vessel (a glass or jug), a tone is heard which is louder the nearer the margin, and the more forcibly, the stroke is delivered. This sound is *exquisitely tympanitic*, and retains the same pitch whether we percuss forcibly or gently, with a narrow or a broad pleximeter. On performing this experiment with different glasses the tone will be found to vary in pitch with the breadth and depth of each vessel, *being high in direct proportion to the shallowness of the glass and the width of its mouth.* The tympanitic character is given to the tone by the vibration of the column of air within the glass and the regular reflection of the sonorous waves from its smooth inner surface.

The conditions presented by the larynx, stomach, and bowel, are exactly similar to those detailed above; we have here to deal with a body of air set in vibration by the percussion-stroke, and enclosed in a cavity whose walls reflect the waves of sound with perfect uniformity. That the tympanicity of the intestinal percussion-note is due simply to the vibration of the air contained in the bowel may be proved negatively. If the whole bowel, or a portion of it, be removed from the abdomen and fully inflated, a ligature being placed on each end, the sound it gives to percussion is no longer tympanitic; the tense intestine becomes capable of entering into simultaneous vibration with the air it surrounds when the shock of the blow is communicated to it; but the vibrations of a membrane (that is, of a *solid* body), being different in kind and extent from those of the intestinal gases, the different sonorous waves interfere with each other, become broken and irregular, and constitute merely a *sound*,—not a *tone* in the physical sense. But as soon as a portion of the air is allowed to escape, the relaxed state of the bowel deprives it of the power of vibrating, and the sound again takes on the tympanitic character.

The alteration in the intestinal percussion-sound thus brought about by *artificial* means finds its parallel in the change which accompanies pathological *distension* of the *bowel* within the abdomen *by gas*; in cases of meteorism of the intestines tympanicity disappears, the percussion-sound over the abdomen becomes louder and deeper in pitch, but not tympanitic.

The physical cause of the normal tympanicity of the intestines at once explains also the non-tympanicity of the normal thoracic sound. The lung in the thorax being always during life slightly over-distended, not only the pulmonary air but the pulmonary tissue also vibrates in response to the percussion-stroke; the sonorous undulations originating in the tissues of the lung mingle with and disturb those of the air, just as the vibrations of the tense intestinal wall obscure those of the intestinal gases, so that no *tone* is given out, but only a *sound* of a non-musical character. This explanation is supported by the fact that the lung, when *relaxed* and reduced to its normal size by removal from the body, yields a note of *tympanitic* quality, only the *air* within it, and *not* the *lung-tissue*, being thrown into vibration; and if the lung be now inflated the tympanitic tone disappears and is replaced by the ordinary percussion-sound of the lung while it is still within the thoracic cavity.

This simple phenomenon—that the lung when raised from the thorax and shrunk to its normal volume gives a tympanitic sound, the distended lung a non-tympanitic sound, to percussion—has been the subject of numerous controversies. Does the tympanicity of the sound depend on the vibration of the pulmonary air? or of the pulmonary substance? or of both combined? These are questions to which very different answers have been given by Wintrich, Mazonn, Körner, Hoppe, Geigel, Schweigger, &c.

Wintrich holds that the tympanitic sound given by the shrunken lung in the dead body originates in the pulmonary parenchyma, and not in the air contained in the alveoli or bronchi; the pulmonary vesicles are too small, the smallest body of air which can give rise to a tympanitic tone having, according to this author, a height of at least six lines, and the pulmonary sound also is somewhat higher in pitch, even than that obtainable from a column of air six lines high; neither can the air in the bronchi be considered as the cause of the tympanitic sound, as narrowing or dilatation of the principal bronchus, or even closure of the same by ligature, makes no change on the pitch of the note,—an effect, however, which is invariably produced by similarly altering the conditions of a column of air contained in an open cavity (see p. 91). Rosenbach, on the other hand, states that such a change *does* follow closure of the principal bronchus; that in these circumstances the sound becomes to a corresponding extent deeper.

The tympanicity of the percussion-sound of the collapsed lung in the dead body can be satisfactorily accounted for, not by regarding the pulmonary cells as columns of air surrounded by membranous walls, and each one capable independently of entering into vibration,—their small size forbidding such an assumption,—but rather by considering them as *continuous*, forming *one large resonant cavity* subdivided by very thin membranous septa which are everywhere of homogeneous structure. *These septa, like the relaxed walls of the intestine or stomach, can indeed reflect sound, but are themselves unable to enter into vibration, as they lack the principal condition necessary to the performance of this function,—a sufficient degree of tension;* it is thus physically impossible to cause them to vibrate. So soon, however, as, by the inflation of the lung, this condition is realised, the tympanitic quality of the percussion-sound is lost, because now not merely the pulmonary air, but also the tense pulmonary tissue, responds to the percussion-stroke, and the substance of the lung, being a *solid body*, naturally gives vibrations quite different in nature from those of the *air* in the alveoli. The result is that these dissimilar sonorous waves to a certain extent neutralize each other and render the production of a tympanitic or musical tone impossible.*

The percussion-sound of the thorax is *tympanitic* in the following pathological conditions:

1. In cases of *excavation of the lung substance;*
2. In cases of *accumulation of air (gas) in the pleural cavity;*
3. When the *tension of the lung tissue is diminished.*

1. TYMPANITIC PERCUSSION-SOUND IN PULMONARY CAVITIES.

Pulmonary excavations, being invariably in direct communication with the bronchi, always contain air, and thus form resonant or reverberating chambers similar to the glass used in the experiment already described. To the production of the true tympanitic sound, however, certain conditions are necessary:

a. This air-space must be surrounded by *firm* walls, capable of reflecting waves of sound. The condensed tissue in which vomicæ are usually enclosed constitutes a solid wall of this character; in the absence of this induration, which is comparatively rare, no tympanitic sound is heard.

b. The cavity must be of a certain size,—as large as a pigeon's egg at least,—and be situated quite close to the surface of the lung; over deep-seated cavities, even when they are of much greater dimensions than above indicated, the percussion-sound has no tympanitic quality. Large vomicæ render a tone which is the more clearly tympanitic the more superficial they are and the thinner the thoracic parietes; cavities, therefore, in the upper lobes of the lungs, in which region the chest-wall—which suffers also in the general emaciation connected with the original disease—is naturally thinnest, give a more decidedly tympanitic percussion-note than those occurring in the lower lobes, chiefly on account of the greater thickness of the layer of muscles covering the latter on the lower part of the dorsal aspect of the chest.

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* Although *tympanicity* is more nearly related to the physical idea of a *tone* than to that of a *sound*, the latter term (as in the expression *tympanitic sound*) being the one best known and longest in use, is retained in the following pages.

The tympanitic character of cavernous resonance is never so intense as that of the intestinal percussion-sound, as it originates in the vibration of a much smaller body of air. It may be either muffled or clear,—muffled when the quantity of fluid in the cavity is greater than the volume of air, clear when these conditions are reversed.

There is one very important sign which is, with very few exceptions (see pp. 96 and 99), associated only with the tympanicity dependent on *pulmonary cavern*,—provided always that the cavity communicates freely with one of the larger bronchi; that is that the *pitch* of the percussion-sound is *higher when the mouth is open, lower when the mouth is shut,* and lower still when the nostrils also are closed (Wintrich). The cause of this modification of the note lies obviously in the variation in the size of the external orifice of the cavity, as it has been already shown (p. 91) that the pitch of the sound given by an air-space which is not completely closed depends not merely on the length of the column of air in vibration, but also on the width of the opening by which it joins the outer air; thus, the tone rises in pitch when the outer orifice is enlarged, and falls when it is contracted, the length of the column of air remaining the same. A similar change can also be demonstrated by percussing on the side of the larynx or on the cheek, the sound becoming lower when the mouth is shut and higher when it is open. It has been observed further that the tympanitic cavernous sound is raised in pitch by full inspiration (Friedreich); this is not wholly attributable to dilatation of the glottis, as the inspiratory rise in pitch takes place even when the bronchus into which the cavity opens is blocked up by mucus,—a condition which is recognised by the disappearance of the sign described by Wintrich, the modification of the percussion-sound by opening and closing the mouth; it proceeds rather from the increase in the tension of the chest-wall, which takes place when the lung expands (see p. 87). In expiration, on the other hand, the tympanitic sound becomes deeper; or it may almost entirely disappear, giving place to a muffled or less clear sound, especially during an attack of coughing. In the latter case the air in the cavities is compressed and for the most part driven into the bronchi by the great augmentation of the intrathoracic pressure, so that the only vibrations which are appreciable are those of the condensed lung tissue.

When the excavations are of very large size, so disposed that their *long diameter* corresponds with that of the lung and of the *body* as a whole, containing both air and fluid, it is sometimes observed that the *intensity* of the tympanitic sound is modified by changing the *position* of the patient; when he is standing or sitting the sound is of a dull tympanitic quality inferiorly, of clear tympanitic quality superiorly,—the fluid gravitating naturally to the lower part of the cavity, the air rising to the upper part; in the recumbent posture the tympanitic note becomes everywhere clear, owing to the uniform distribution of the fluid over the entire posterior surface of the cavity. Not only the intensity but also the pitch of the percussion-sound of large excavations is altered when the position of the body is changed; when the cavity is of such a form and so situated that its long diameter is directed from above downwards the tone becomes higher when the patient assumes the sitting posture, as in this way the vibrating column of air is considerably shortened; when the cavity is deepest antero-posteriorly the pitch of the percussion-note is lowered in the upright position (Gerhardt). The last-mentioned sign, when unmistakeably present, is absolutely pathognomonic of the existence of cavity, because under physiological conditions a deepening of the pitch of the percussion-sound is never produced by raising the body from the recumbent to the erect posture; the occurrence of the former phenomenon, however, the elevation of pitch in the sitting posture is less conclusive, as it may also be determined by the increased tension of the thoracic parietes (see p. 87).

Gerhardt also believes that the dimensions of pulmonary vomicæ may be definitely ascertained by means of Helmholtz's *resonators*. If a suitable resonator be held before the patient's mouth, which should be open, the tympanitic cavernous percussion-note is greatly increased in intensity; and as the size of the cavern and that of the resonator which produces this effect stand in direct proportion to each other the diameter of the one may be taken to represent that of the other. The researches of Eichhorst and H. Jacobson do not confirm these statements; it was found on post-mortem examination that the actual size of the cavities differed very considerably from the estimate formed by means of the resonators, being sometimes three, five, or more times larger or smaller than had been expected. It was further shown that the same resonators which, when held before the open mouth, intensified the tympanitic cavernous sound, rendered more clear also the percussion-sound on the healthy side of the chest. This method of examination, therefore, cannot be depended on to give trustworthy results.

2. The Tympanitic Percussion-sound in Pneumothorax.

In pneumothorax the air with which the pleural cavity is filled is caused to vibrate by the percussion-stroke, while the waves of sound are regularly reflected by the chest-walls; the same conditions are thus provided for the production of the tympanitic sound as in percussing the larynx or the glass used in the experiment formerly described, (p. 91). The area rendered tympanitic varies in extent, being large or small according to the greater or less amount of compression of the lung which has taken place. Opening or shutting the mouth has no effect on the pitch of the sound, the cavity occupied by the air being a closed one; enlargement or diminution of the vibrating column of air therefore, in the sense in which these changes occur in pulmonary cavities which are in free communication with bronchi, is out of the question. It is only in very rare cases, in which the fistula through which the air has entered the pleural sac continues patent and is of sufficient size, that it is possible to drive a certain quantity of air from the cavity of the pleura with each percussion-stroke: in these cases the pitch of the sound may be varied by opening or closing the mouth.

Tympanicity in pneumothorax persists only so long as the tension of the imprisoned air continues moderate in degree. If the bronchial fistula or the rupture in the wall of the pulmonary cavern (the latter being the most common cause of pneumothorax) be not immediately closed by the products of adhesive inflammation, air rushes into the pleura at each inspiration, till the lung is reduced to a state of complete collapse [*]; the tension of the air in the affected side of the chest thus becomes so great that the tympanitic quality is lost from the percussion-sound and is replaced by the metallic or amphoric sound to be subsequently discussed.

The irritation consequent on the admission of air into the pleura usually provokes an attack of inflammation in that structure, followed by more or less copious effusion; the fluid, as in most cases, may not rise above the lower and posterior part of the cavity, or it may encroach on it to a still greater extent, constituting the condition known as pyo-pneumothorax; it may even completely fill one side of the chest (pyothorax) and in that way bring about the cure of the pneumothorax. In the latter case the percussion-sound on the affected side is absolutely dull, in the former cases it continues tympanitic above the level of the exudation. In pyo-pneumothorax also *change in posture developes a change in the character of the sound*, as in each position in which the body may be placed the fluid invariably seeks the lower level, the air the higher. Thus the dull sound of the anterior and lower part of the chest in the standing or sitting position becomes at once tympanitic as the patient assumes the recumbent posture, that of the one side of the chest being similarly modified when decubitus is on the opposite side, that of the lower and posterior parts of the thorax when the patient lies prone on the abdomen. Should the pleuræ at any point have formed adhesions before the occurrence of the pyo-pneumothorax the air is excluded from certain parts of the cavity, and encysted pyo-pneumothorax is the result; in such cases change of attitude is attended by no modification of the physical signs.

3. TYMPANITIC PERCUSSION-SOUND DUE TO DIMINISHED TENSION OF THE LUNG SUBSTANCE.

Those conditions which favour the *retraction* of a greater or less portion of lung, (that is, its return to its dimensions in the relaxed state in which it is found when respiration has ceased, the pulmonary tissue being always more or less on the stretch during life), give rise very frequently to a tympanitic percussion-sound,—just as it occurs in the lung when removed from the thorax and so allowed to resume its *natural* volume.

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[*] The air forced into the pleural sac during inspiration does not, or does to but a very slight amount, escape during expiration, as the pulmonary fistula is closed valve-fashion by the increasing pressure of the air in the cavity.

Amongst these conditions are: *a.* Pleurisy, *b.* Pneumonia, *c.* Œdema of the lungs, *d.* Caseous pneumonic degeneration in the upper lobes.

a. In *exudative pleurisy* the lung shrinks to a degree proportionate to the amount of the exudation, and it consequently loses to the same extent in tension: when this retraction reaches a certain point, which is not the same in every case, the percussion-sound takes on the tympanitic quality. Even at the beginning of the exudative process, when the layer of effusion is not yet of sufficient thickness to give rise to percussion-dulness, the sound at the posterior and lower part of the thorax is often found to be of a dull tympanitic character; this speedily disappears with the augmentation of the exudation, and the parts become completely non-resonant.

In pleurisy with moderate effusion, the tympanitic sound is observed *above* the level of the fluid, in the anterior and lateral parts of the thorax. The tympanitic quality is more or less distinctly appreciable according to the degree to which the free portions of the lungs are reduced in volume; it is lost as the exudation increases, and reappears at the beginning of the stage of absorption when the lungs commence to expand again.

But not in every case of pleuritic effusion is the sound tympanitic above the region occupied by fluid: it is often merely *deepened in pitch*, and sometimes even shows no departure from the normal standard (see p. 89 *et seq.*). The causes of these differences in apparently identical physical conditions, so far as the exudation alone is concerned, should probably be sought in the unequal tension of the lung-tissue in different individuals. A greater degree of tension is necessary for the development of a tympanitic sound than for the production of one which is abnormally low in pitch but non-tympanitic. In some cases, in which the percussion-note above the effusion undergoes no change, a decrease in the elasticity of the lungs may be shown to exist, rendering any great degree of retraction impossible,—in chronic bronchial catarrh, for instance, especially when associated with vesicular emphysema: at other times there are other causes, such as a more rigid condition of the thoracic parietes, to account for this phenomenon.

It was Skoda who first drew particular attention to the occurrence of tympanicity in cases of pleuritic exudation, and satisfactorily made out the nature of the physical conditions on which it depends, though these seem to have been not quite unknown even to Auenbrugger. This sound is sometimes named the "bruit scodique" by French physicians.

b. A *tympanitic percussion-sound* is sometimes also heard in *pneumonia*, especially in the first stage and in that of resolution, though it is not unusual to find that in the stage of hepatization the permeable parts of the lung in the immediate neighbourhood of those which are completely consolidated give a sound of this character; it is further more common in the upper than in the lower lobes. Its cause may be of a twofold nature. Thus, when the *superior* lobe is hepatized a tone of tympanitic quality, arising from the vibration of the column of air contained within the principal bronchus of that lobe, may be elicited by forcible percussion; in these circumstances also the sound, like that of pulmonary cavern and of the larynx, is observed to vary in pitch on opening and closing the mouth. In hepatization of the *lower* lobes, on the other hand, where there are no large bronchi, the cause of the tympanicity is evidently loss of tension in the lung substance; the lung shrinks somewhat in the vicinity of hepatized parts, these latter being rendered abnormally large by the infiltration of the alveoli. In the first and third stages of pneumonia the relaxation of the pulmonary tissue is due to infiltration of the air-cells, caused by engorgement of the capillaries (first stage) or by the presence of fluid and air in their interior (third stage). The tympanicity of the first stage merges gradually or rapidly into absolute dulness as the process of hepatization begins; in the third stage it slowly passes off, giving place to the normal sound when the exudation has been removed from the alveoli by absorption.

c. The occurrence of the *tympanitic percussion-sound in œdema of the lungs*, in which the pulmonary vesicles are filled with transuded fluid and air, may be explained in the same way: the lung-substance loses in tension, the quantity of air contained in the alveoli being smaller.

This variety of tympanicity may be reproduced after death by forcing fluid through the trachea into the vesicles and at the same time *inflating* the lungs; (inflation is an absolutely necessary part of the proceeding, as the non-distended lung gives *naturally* a tympanitic sound).

d. The percussion-sound is frequently observed to be *tympanitic* in the supra- and infra-clavicular regions when the summits of the lungs become the seat of *caseous pneumonic infiltration ;* in such cases it is always somewhat muffled and does not change its pitch when the mouth is opened or shut,—features by which it may be distinguished from the tympanitic sound of pulmonary *cavity* in the apices. It is produced only when the infiltrated tissue has not yet been completely deprived of air, or when small scattered patches of permeable tissue are still found between the portions which have become consolidated. Here also the tympanicity depends on the relaxation of the lung-substance and diminution of its air-contents. Infiltration of the apices specially favours the development of the tympanitic tone, as the chest-wall over these parts is thin and becomes still thinner as emaciation goes on.

There is a special form of tympanicity which is observed in certain cases of condensation of the lung, most markedly in the first and second intercostal spaces and usually on the *left* side; this is

THE TRACHEAL RESONANCE OF WILLIAMS.

In percussing the trachea a tympanitic sound is obtained, which, like that given by pulmonary cavities, rises in pitch on opening the mouth and falls on shutting it, and which becomes still lower when the nostrils also are closed. This sound is lost at a point on the surface corresponding to the bifurcation of the trachea, being overpowered by that from the tissue which covers the principal bronchus. When, however, the upper lobe is completely consolidated this predominance of the ordinary pulmonary percussion-sound ceases, and the tympanitic sound from the column of air in the bronchus is appreciable on striking the front of the chest somewhat forcibly. The tone, like that furnished by excavations in direct communication with the trachea, becomes higher in pitch and louder on opening the mouth, and considerably deeper on closing it.

This tracheal resonance, therefore, is most usually observed when the upper lobe of the left lung is completely solidified by pneumonic *infiltration*, and occasionally also when this takes place from other causes,—when, for example, the lung remains in a state of collapse after the absorption of pleuritic exudation, and in those rare instances of *encysted* pleuritic exudation situated at the upper and anterior part of the left side of the chest; it is met with also in some cases of pleurisy in which the effusion is not confined by adhesions, when the quantity of fluid is not so great as to separate the lung too much from the front of the chest. The ribs, being somewhat flexible toward their sternal ends, yield to the force of the percussion-stroke, and in that way favour the production of the sound, whilst at the corresponding part of the posterior surface of the thorax this sign may be entirely wanting, owing to the greater thickness of the layer of muscles and to the rigidity of the chest-wall in that region. That this treacheal resonance is found almost exclusively on the left side is probably due to the greater length of the principal bronchus on that side.

Skoda regards the tracheal resonance of Williams as identical with the tympanitic sound of the retracted lung in pleurisy; this view, however, is opposed by the facts that it is audible in complete hepatization of the upper lobe, where no retraction exists, and that the pitch of the sound is subject to the usual variations on opening and closing the mouth, which is not the case with the true "bruit scodique."

To these three qualities of the percussion-sound Skoda added a fourth, *fulness* and *scantiness* of the sound. These terms are still frequently used by scientific writers and are generally considered as synonymous with loudness and dulness, though Skoda gave to them quite another signification. He intended them to bear reference to the size of the vibrating body, as is indicated by his citing, as an illustration of his meaning, the difference in the sounds given by bells of different sizes: he says, "the faintest sound from a large bell, or the loudest ringing of a small one, conveys to us at once the idea of the size of the bell in vibration,—the former sounds *full*, the latter *scanty*."

This fourth quality, which is still defended by Skoda, is now, on physical grounds, almost universally rejected. The following are the principal objections urged against it by various authors (Mazonn, Schweigger, Wintrich, &c.):—that which in music is designated as *fulness* coincides sometimes with the *intensity*, sometimes with the *pitch*, of a sound; the sound of a large bell is invariably deeper than that of a small one, and is louder also even when both are struck with equal force. This difference does not depend on the volume of the resonant chamber, but on the material of which its walls are composed; a small violin may yield a fuller sound than a large one. Similarly in the human voice difference in sonorousness does not arise from difference in the size of the vocal cords. Even the examples quoted by Skoda in support of his theory regarding this quality of percussion-sound in the human chest and abdomen show that *fulness* is identical with loudness and depth, *scantiness* with dulness and elevation of pitch. Thus, if an entire lobe of the lung be hepatized, with the exception of a small spot situated near the surface, this isolated and permeable patch of tissue gives, according to Skoda, a sound which is indeed clear, but which is also scanty, inasmuch as only a very small body of air is caused to vibrate, whilst the rest of the pulmonary substance is incapable of entering into vibration; in the classification we adopt the percussion-sound in such a case is regarded as neither so loud nor so deep as that of the healthy lung, but as somewhat duller and higher in pitch. On the other hand, when the air has free access to all parts of the lung with the exception of a small superficial patch, the sound obtained over this hepatized spot is, according to Skoda, somewhat duller but still moderately full, as only a very small portion of lung is rendered unsusceptible of vibration; in our terminology, however, such a sound is described as less intense (muffled) but still moderately deep. In the same way the full sound rendered by the stomach is considered loud and deep, and the scantier intestinal sound is spoken of as being higher and less intense. The opposite of the full sound, the typically "scanty" sound, is absolutely indistinguishable from the perfectly dull sound. As the fulness and scantiness of the percussion-sound are traced to the longer or shorter duration of the vibrations of the resonant body, the scanty sound has also been named the *short or shorter sound*, a term which seems inappropriate, as its opposite, the *longer sound*, has never been admitted into the nomenclature of percussion; *muffled* or *obscured* would be a more fitting term.

Two qualities of sound occasionally met with still demand consideration, the *cracked-pot sound* and the *amphoric* or *metallic resonance*.

THE CRACKED-POT SOUND.*

(Bruit de pot fêlé).

This is a hissing or rattling noise, which may be imitated in two ways: by percussing with the hammer and pleximeter, applying the latter somewhat loosely to the chest-wall, so that beneath it is left an interspace containing air, though in this case the sound produced is wanting in the tympanitic or metallic quality;

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* Laennec was the first who employed this expression. Baas has more recently described it as the [illegible] sound.

and by laying the palms of the hands on each other crosswise, in such a way as to form a hollow chamber, and then striking the back of one of them against the knee. In the latter case the concussion produces a noise which is exactly like the chinking of money, a sound which well-marked bruit de pot fêlé simulates exceedingly closely. In both of the experiments above described the sound is caused by the *sudden escape* or discharge of *air subjected to pressure*. In health the cracked-pot sound may be elicited by percussing forcibly the chest of children when screaming, or that of adults when singing a long-sustained note, the air being thus suddenly compressed by the force of the percussion-stroke and made to rush out with a hissing sound.

When the chest is plentifully covered by hair this sound is not unfrequently heard, especially over the sternum; here it is caused by the presence of a small quantity of air which lodges in the meshes of the crisp wiry hair which grows in this region, and by its sudden escape when the percussion-stroke is delivered. It disappears on moistening the parts, the hair then lying flat and close to the surface of the chest.

The cracked-pot sound occurs in the following pathological conditions:

1. When there are *cavities of moderate size in the lung-substance*, communicating freely with a bronchus of medium calibre, situated superficially and at parts over which the chest-wall is sufficiently yielding and the covering of soft tissues has been rendered thin by emaciation. It is heard almost exclusively at the upper part of the *anterior* aspect of the thorax, from the first to the fourth rib, more especially in the infraclavicular region, being more distinctly audible when percussion is practised during expiration, and *most clearly when the patient keeps his mouth widely open*. After repeated examination, or accidental closure of the bronchus leading to the cavity, the bruit disappears, to return as soon as the cavity is refilled with air or the superabundant secretion of mucus has been expelled by coughing. To produce this sound a firm blow is necessary, so that it is better to use the hammer than the finger.—When connected with vomicæ in the lungs the percussion-note is further always of tympanitic or metallic quality, which, indeed, is the element from which it derives its "chinking" character; this tympanitic tone becomes inaudible at the instant in which the hissing sound is produced by the rush of air from the relatively wide

cavity into the relatively narrow bronchus, when the regular reflection of the sonorous waves, which is necessary to the formation of a tone, is interrupted. Should the cavern contain much fluid it also is agitated by the shock of percussion, and something resembling a râle is heard mingled with the cracked-pot sound.

2. In some cases of *pleurisy*, and at those parts of the lung, situated above the level of the fluid, to which the air still has access. The condition of the pulmonary vesicles, retracted and approximated to each other on account of the diminution of their air-contents, seems here to favour the escape of the air on percussion.—The bruit de pot fêlé sometimes observed in the vicinity of *pericardial exudation* (Leichtenstern) originates also in the retraction of the pulmonary substance compressed by the effusion.

3. Occasionally in cases of *pneumonia*, over those permeable and relaxed parts of the lung immediately adjoining those which are hepatized. The cause of the phenomenon is here the same as in pleurisy. Sometimes, however, it is heard also over the condensed portion; this, as in the case of a cavity containing air, can arise only from the sudden expulsion of air from one of the larger bronchi leading to the affected part.—At other times both causes, relaxation of the lung-tissue and concussion of the air in the bronchi, are in operation in pneumonia (Löb).—In pleurisy and pneumonia the cracked-pot sound does not become louder on opening the mouth.

4. In cases of *thoracic fistula*, such as those which sometimes occur after paracentesis thoracis. The air in the pleural sac escapes through the fistulous opening with a *hissing* noise on percussing in the immediate neighbourhood of the orifice; when the latter is closed the cracked-pot sound is not developed.—The same explanation is applicable in some instances of *pneumothorax* from stabbing or gunshot wounds.—In certain rare cases of pneumothorax from internal causes, in which the pleural cavity is in direct communication with a large bronchial fistula (Oppolzer, Rollet), the bruit de pot fêlé may be heard.

The cracked-pot sound is, as the foregoing facts indicate, always dependent on the same causes, though the particular conditions in which it is found may be very different anatomically; it is most frequently due to the presence of vomicæ in the upper lobes of the lungs, the cases of pleurisy, pneumonia, and pneumothorax in which it is observed constituting a very small fraction of the total number met with. The occurrence of the sound, therefore, in the course of a pulmonary affection known to be phthisical, may be taken as absolute proof of the formation of *cavity*.

AMPHORIC RESONANCE.

(Metallic percussion-sound, metallic echo.)

This sound is identical with that obtained on striking on the side of an empty cask, a pitcher, or large india-rubber ball, &c.

On the whole, it most closely approaches in quality the tympanitic note, differing from it in being somewhat metallic in timbre, by being higher in pitch (its fundamental tone, which is variable, being also accompanied by overtones, or segmental tones), and by being of longer duration. The ordinary tympanitic sound ceases at once after the percussion-stroke; this amphoric resonance (the metallic "klang"), however, lasts some time longer, as the higher overtones die away much more slowly. This modification of the tympanitic sound by the addition of a metallic echo may easily be studied in one's own person: the cheeks, when relaxed, the mouth being closed, give a sound which is simply tympanitic, when powerfully distended a sound of amphoric character.

The examples cited above show that amphoric resonance is produced in *large air-filled caverns, surrounded by smooth walls* which regularly and uniformly reflect the waves of sound, completely enclosed on all sides or communicating with the external air by means of a narrow opening. Apart from the force of the percussion-stroke, the intensity of the sound depends chiefly on the size of the cavity; an audibly metallic note is obtainable only from a chamber whose transverse diameter is at least 3—4 cmtr., as has been proved by Merbach and Leichtenstern in a series of experiments with cylindrical vessels having tense elastic walls. The pitch of the metallic tone in vomicæ which are not spherical in shape varies with the length of the longest diameter; percussion in the direction of the shorter diameter gives a higher note, in the direction of the long diameter a lower note.

In the thorax amphoric resonance is heard over *large pulmonary cavities*, and in cases of *accumulation of gas in the pleural sac*.

Excavations in the lungs must have a length of at least 6 cmtr.

of the percussion-sound is impossible without a knowledge of the normal pulmonary sound, of the boundaries of the areas over which it is audible, and of the physiological differences in its intensity and pitch at various points on the surface of the thorax. In the practice of percussion, therefore, the study of the normal sound must precede that of its pathological modifications; but the didactic representation of the results of topographical percussion, on the contrary, can be properly understood only after the causes and diagnostic significance of the different qualities of percussion-sound and its pathological alterations have been discussed.—Besides the ordinary anatomical descriptive terms, such as supra- and infra-clavicular, supra- and infra-spinous regions, &c., the ribs and intercostal spaces, the sternum and vertebral column, are all made available in topographical nomenclature. The numbering of the ribs starts at the first,—when it cannot be distinctly felt, at the second, the sternal insertion of which stands out prominently. The number of any of the lower ribs is best determined by counting from below upwards, the twelfth being always easily made out.—As aids to more exact topographical description the anterior, lateral, and posterior surfaces of each side of the thorax are divided by vertical lines drawn at nearly equal distances from each other: the *median line* passes vertically through the middle of the sternum, the *sternal line* parallel with it at the edge of the bone, *the mammillary line* through the nipple, the *parasternal line* midway between the two last-mentioned lines, the *middle axillary line* through the middle of the axilla (in front of and behind this being the *anterior* and *posterior* axillary lines), the *scapular line* perpendicularly through the inferior angle of the shoulder-blade.

1. THE NORMAL LIMITS OF THE LUNG.

a. *The upper limit.* The lungs on both sides rise anteriorly 3 to 5 cmtr. above the clavicles, occupying there a triangular space the outer side of which is formed by the free edge of the trapezius muscle, the inner by the clavicular portion of the sternomastoid, the base by the clavicle. Posteriorly the apex of the lung occupies an area bounded externally by the trapezius muscle, inferiorly by the spine of the scapula, superiorly by the spinous process of the seventh cervical vertebra.

b. *The anterior* (*inner*) *limit* follows the line of the anterior inner margins of the lungs. These, passing downwards from the apices, approach each other and meet behind the sternum at the level of the second rib, and remain in apposition, separated only by the anterior mediastinum, as far as the level of the fourth rib; beyond that point they diverge, the left, after turning abruptly outwards, inclines again slightly towards the sternum behind the cartilage of the fifth rib, and ends, opposite the sixth costal cartilage, in the left lower margin; the right runs nearly perpendicularly behind the sternum from the level of the fourth rib to the sixth costal cartilage, where it joins the inferior border at almost a right angle.

c. *The inferior limit* is formed by the lower borders of the lungs. When the diaphragm is in a moderate state of contraction, as during quiet respiration, the inferior margin of the *right* lung is found at the upper edge of the sixth rib in the parasternal and mammillary lines, at the upper edge of the eighth rib in the axillary line, at the ninth rib in the scapular line, and at the tenth rib close to the vertebral column. The lower margin of the *left* lung is situated at the lower border of the sixth rib in the mammillary line, at the upper border of the eighth rib or in the eighth intercostal space in the axillary line, at the ninth rib in the scapular line, and at the tenth rib close to the vertebral column.—In aged persons the pulmonary boundaries are about a rib's breadth deeper, in children about the same distance higher.

The lungs, when expanded in inspiration, pass over these limits, especially inferiorly and anteriorly, least of all in the upward direction. In ordinary circumstances, when respiration is slow and tranquil, any extension of the boundaries of the lung which may take place is but trifling, almost imperceptible, the displacement at the right lower border, in the mammillary line, amounting only to about 1 cmtr. When inspiration is forced, on the other hand, the lower edge of the right lung may sink to the extent of 3 cmtr. in the parasternal and mammillary lines, or even as much as 4 cmtr. in the axillary line; the ascent of the upper part of the lung varies from ½ cmtr. when breathing is quiet to 1½ cmtr. on making a full inspiration. Change of posture also gives rise to some displacement of the margin of the lung; on turning from the back to the side while in the recumbent position the lung which is uppermost descends usually about 3 cmtr. in the axillary line, and about 2 cmtr. in the mammillary line.

For diagnostic purposes it is of the greatest importance to define accurately, by means of percussion, the lower border of the right lung, inasmuch as if it be shown that it does not go beyond its normal limits the possibility of the existence of one of the most common lung diseases, vesicular emphysema, is excluded. This, fortunately, is easily accomplished, as the lung, anteriorly and laterally, is separated from the liver merely by the thickness of the diaphragm, so that a marked difference between the clear pulmonary sound and the dull hepatic sound is at once perceived. The line of demarcation of the lower margin of the right lung is thus identical with that of the upper edge of the liver.

The delimitation of the right lung inferiorly affords an excellent opportunity of studying the gradual transition of the percussion-sound from perfect resonance to absolute dulness. In the first place, during quiet respiration or, still better, after forced expiration, the upper boundary of the *absolutely* dull hepatic area in the mammillary line should be defined and marked on the skin with black dermographic crayon (crayon lithographique), when it will usually be found to coincide with the lower edge of the sixth rib. In the same way the lowest point at which the pulmonary-sound is still perfectly clear, which is generally about the level of the fifth rib, is to be ascertained and shown on the surface. The region lying between these two points is about an inch in depth, and constitutes the zone of transition from resonance to dulness; in it the percussion-note is somewhat muffled or obscured. If this part be further examined by percussing with one finger on the other the definite boundary line between lung and liver is found, as a rule, at the upper edge of the sixth rib. The same method of procedure is adopted in delimiting the lung in the parasternal and axillary lines. The extreme accuracy and trustworthiness of the results so obtained have frequently been shown on the dead body, by inserting long needles into the tissues along the lines indicated by physical examination.

It is of importance from a diagnostic point of view also to ascertain the *mobility of the lower border of the lungs*. As in full inspiration the lung stretches downwards to a point 2 to 3 ctmr. lower than in expiration, the upper part of the area which in the latter condition gives a dull liver-sound, is in the former case perfectly clear and resonant: this change, when present, proves the complete mobility of the lung. When this rising and falling movement of the margin of the lungs is wanting, as in cases in which the visceral and costal pleuræ are extensively adherent, the percussion-sound of the region in question remains unaltered in both phases of respiration. In severe cases of pulmonary emphysema, also, the edge of the lung is nearly or quite motionless in inspiration; here, therefore, percussion reveals to us the fact that the alveoli of the lower part of the lung are no longer elastic, but have lost their capability of expansion.

To determine the *upper* margin of the lung, the highest point at which the clear pulmonary percussion-sound is appreciable should be noted during expiration; if the apex of the lung be expansible the clear area extends upwards during inspiration, but if, as so frequently happens, it be the seat of cheesy degeneration, the clear percussion-area is lower in point of position even during expiration (Seitz) and rises but slightly or not at all during inspiration. The difference in the extent of the movement at the apices on the healthy and the affected sides, measured with Haenisch's stethograph, varies from ¼ to a little over ½ ctmr. If the percussion-sound be absolutely dull, the apex being completely consolidated and void of air, inspiration does not make it clearer. When the apex of one lung is affected, however, the range of movement possible on the healthy side is also generally somewhat restricted.

The movements of the *anterior inner* borders of the lungs may be traced by the percussion-sound becoming louder (clearer) over the sternum and in the cardiac region during inspiration. In deep inspiration the edges of the lungs creep forward, so that the greater part of the heart is covered and the cardiac dulness almost entirely masked.

When the *anterior* border of the *left* lung is pathologically distended (by emphysema) the cardiac dulness is diminished in area or completely obliterated, and the sound over the heart is loud and clear; if, on the other hand, it be adherent to the costal pleura, incapable of expansion or forward movement, the area of cardiac dulness is unaltered either in inspiration or expiration. When this part of the left lung atrophies it retreats, the base of the heart comes to a large extent into contact with the chest-wall, the pulsation of the heart is seen and felt as far upwards as the third or fourth intercostal space, and the region of cardiac dulness is enlarged. The mobility of the anterior margin of the *right* lung is recognised by the greater clearness of the sternal percussion-sound in inspiration.

The movements of the *posterior upper and lower* borders of the lungs, also, are well-marked and easily made out by the extension of the clear percussion-sound during inspiration.

Pathologically the *posterior upper* margins of the lungs recede in contraction of the apices, while the *posterior lower* borders extend downwards to the eleventh or twelfth rib in pulmonary emphysema.

The position of the lobes of the lungs, with relation to the thoracic wall, is as follows:—

Right lung (three lobes).—The *upper* lobe reaches downwards on the front of the chest to the fourth or fifth rib, laterally to the fourth rib, posteriorly to the spine of the scapula; the *lower* lobe lies between the spine of the scapula and the tenth rib posteriorly, and laterally between the sixth and the eighth ribs. Between the upper and lower lobes comes the *middle* lobe, which laterally occupies the space included between the fourth and sixth ribs, and anteriorly extends to the lower margin of the lung.

Left lung (two lobes). The *upper* lobe comes down on the front of the chest to the sixth rib in the mammillary line, (to the inside of which point lies the heart), and laterally to the fourth rib; below this, and reaching to the base in front and from the spine of the scapula to the base behind, is the *lower* lobe.

2. Regional Percussion.

On the *right* side of the chest in *front*, from the apex to the fifth intercostal space, the percussion-sound is loud (clear), deep, and non-tympanitic; resonance is greatest from the clavicle to the fourth rib,—the clavicle itself giving a note scarcely less clear than that of the supraclavicular region,—and least at the apex (on account of the smallness in volume of the subjacent portion of lung) and in the fifth intercostal space (owing to the thinning of the lung at this point and its proximity to the liver). From the sixth rib to the margin of the thorax the sound is absolutely dull; but about the lower edge of the liver, more particularly in children, the intestines give to the percussion-sound a more or less distinctly tympanitic quality.

In the neighbourhood of the sternal insertions of the two first ribs on both sides, most often on the right side, the percussion-note is usually less clear than (for example) in the infraclavicular region. This is caused by the thinness of the lung towards the border, the volume of air thrown into vibration being small.

On the *sternum* the sound is clear, deep, and non-tympanitic. On the manubrium sterni it is somewhat less clear than on the body of the bone, but is nevertheless nearly as clear as that obtained at the apices. That the manubrium sterni should be resonant to percussion though there is no portion of the lung-tissue behind it, but only the trachea, the œsophagus, blood-vessels, &c., admits of explanation only on the supposition that its power of entering into vibration is such that the movements excited in it by the blow are at once transferred to the neighbouring lung-tissue. This is what occurs also at the lower part of the sternum,—though here and over the xiphoid cartilage the sound is less loud and clear, owing to the proximity of the right ventricle of the heart and of the left lobe of the liver.

The sternum may be rendered less vibratile if during the examination an assistant press firmly with both hands on the sides of the thorax near the sternum (Mazonn), when the sound over those parts of the bone behind which no part of the lung is situated, becomes decidedly duller.

The percussion-sound is clear on the *left* side in *front*, from the apex of the lung to the upper edge of the fourth rib, and dull from the fourth rib downwards through the cardiac region to the point at which the apex-beat of the heart is perceptible (the fifth intercostal space). On the sixth rib we have the first trace of the tympanitic stomach-sound, which, lower down, about the margin of the thorax, passes into that of the colon.—At the base of the left side of the chest, lying along the anterior lower border of the ribs, is a certain region in all parts of which the percussion-sound is tympanitic; it reaches posteriorly to the ninth or tenth rib, is about 8 or 9 cmtr. in breadth at its broadest part and somewhat crescentic in general conformation (Traube). This space is encroached upon by the lung at each inspiration, when its upper part yields the clear pulmonary sound; and, similarly, it becomes smaller in all pathological conditions (pulmonary emphysema, for example) involving downward displacement of the diaphragm and stomach, and is enlarged in those conditions (atrophy of the left lung, for instance) in which the diaphragm is drawn upwards. It is reduced in size also in cases of effusion into the left pleura, when the fluid is so abundant as to occupy the front of the cavity, the pressure from above pushing the diaphragm, and with it the stomach, downwards; the semilunar shape is then completely lost, and the area formerly resonant is now dull to percussion. The beginning of the process of absorption of the fluid is often announced by the return of an obscurely tympanitic percussion-sound in the region described.

On the *posterior* surface of the chest the pulmonary percussion-sound is heard on both sides as far down as the tenth or eleventh rib; it is less clear than in front, owing to the ample development of the dorsal muscles and the greater resistance offered by the ribs. The sound is least clear in the supra- and infra-spinous regions and at the lower part of the thorax. Bending of the body to one or other side, asymmetry of the shoulders, and frequently the fuller development of the muscles on the right side of the back, give rise to physiological differences in the percussion-note; these, however, may usually be eliminated by directing the patient to assume such a position that the parts on each side are brought into the same condition during examination, particularly by making him cross the arms in front and stoop a little forward; in this position the posterior surface of the chest is alike on both sides and the muscles in an equal state of tension.

On the *right lateral* surface the sound is loud and clear down to the eighth rib, at which point the liver-dulness begins.—On the *left lateral* surface the pulmonary sound may be elicited as far downwards as the ninth rib; between the ninth and eleventh ribs the percussion-note is rendered dull by the spleen, while at the eleventh rib the tympanitic sound of the colon is first detected.

THE SENSE OF RESISTANCE ACCOMPANYING PERCUSSION.

The more solid the consistence of any body the greater is the sense of resistance experienced by the fingers in pressing or percussing upon it; it is from this cause that in ascending a stone stair the resistance perceived by the soles of the feet is greater than in ascending one constructed of wood. Hard bodies, such as wood or stone, offer very considerable resistance to percussion, soft bodies, such as cotton-wool, feathers, &c., almost none. Similar differences are observable, in percussing the organs removed from the thorax and abdomen, between the resistance of a compressible, permeable lung and that of a firm, hepatized lung or any other solid body (liver, heart, spleen, &c.). But this sensation depends not only on the consistence but also on the thickness of the solid body; thus, the thin spleen, removed from the abdominal cavity, is considerably less resistant than the thick liver, while the relatively thin left lobe of the liver gives this feeling to a much less intense degree than the right. —The existence of these varied degrees of resistance may also be demonstrated on the body by simply percussing the lungs, liver, spleen, breast, &c., though the differences will be found to be hardly so well marked as when examining the organs outside the body, as the resistance of the thoracic walls has to be taken into account.

The resistance of the lungs may be *increased* or *diminished;* the former is an exceedingly common condition, the latter somewhat rare.

Increase of the sense of resistance at various points occurs, even though the lungs be normal, from the presence of certain obstacles which the structure of the thorax and the soft parts offer to the proper performance of percussion. Amongst those unfavourable conditions are excessive development of the bones forming the framework of the chest, narrowness of the intercostal spaces, unusual convexity of the ribs, and the presence of a large deposit of fat (especially in and around the female mamma).

The principal pathological change which gives rise to increased resistance in the lung is impermeability of its tissue to air, whether produced by infiltration, atrophy, or compression (by fluid or tumours in the pleural sac); it is thus usually traceable to the same causes which are known to render the percussion-sound dull. Hence increased resistance and a dull percussion-sound are always found associated, and point to the same conclusion—*that the subjacent media are partially or wholly deprived of their capability of entering into vibration.*

Other things being equal the feeling of resistance increases progressively with, though not proportionately to, the diminution in the air-contents of the lung; the completely air-less hepatized lung is thus much more resistant than that which is solidified (but less completely) from other causes; large pleuritic effusion whose existence we infer from alteration in the pitch of the percussion-note, from the presence of the tympanitic sound, from variation in the pitch of the sound on opening or closing the mouth, from the bruit de pot fêlé and the amphoric resonance. The increased sense of resistance, by which dense organs may at once be detected on percussion, is perceptible to but a very slight degree in the phonometric method.—I consider phonometry badly adapted for the examination of the abdomen, except, perhaps, in the delimitation of the liver and spleen. There is almost no difference between the phonometric sound of the thorax and that of the abdomen, though the percussion-notes of these parts are so exceedingly unlike and characteristic; while even the most pronounced pathological changes in the abdomen,—very abundant ascites, for instance,—announce themselves very much less distinctly by phonometry than by percussion. Finally, a phonometric examination of the whole thorax, as may be inferred from the description of it already given, cannot be made in a shorter time than a period at least three times as long as that required for examination by percussion. For all these reasons phonometry,—however interesting it may be scientifically, and however certain it may be that in the hands of those skilled in its use it may suffice for the recognition of very fine shades of difference in resonance,—has hitherto found little favour with practical physicians as a means of physical diagnosis.

AUSCULTATION OF THE LUNGS.

Historical Note.

The knowledge of at least some of the phenomena of auscultation, more particularly of the sign known as *Hippocratic succussion*,[*] observed in pyo-pneumothorax, dates from the time of Hippocrates. That Hippocrates was also acquainted with the friction[†]-sound of pleurisy and many of the catarrhal sounds seems equally unquestionable, not merely from the description of these given in his works, but also from his account of the diseases in which they were heard.

His observations, however, seem soon to have been entirely forgotten, as there are only a few scattered and obscure references to auscultation, but no precise enumeration of signs, in the works of certain of the ancient writers who came after him.

The real discoverer of auscultation, and of almost all its phenomena in the domain of the respiratory and circulatory organs, is *Lænnec* (born 1781, died 1826). The discovery was made in the year 1816, the first stethoscope which Lænnec used, to enable him to hear better the beat of the heart in a case of cardiac disease, being a roll of paper. Three years' further study and observation in the Hôpital Necker made him acquainted with nearly all the auscultatory signs, the result of his labours being first given to the world in 1819, in a work entitled "Traité de l'Auscultation médiate et des maladies des poumons et du cœur."—*Skoda* has submitted Lænnec's teaching to a most searching criticism, and, by tracing each of the auscultatory phenomena to its physical cause, has worked as great a reformation in this department of physical diagnosis as in the science of percussion. He has not only simplified matters by setting aside many of the points which found a place in Lænnec's system, but has also established, on physical principles, a classification of the phenomena of auscultation which has found universal acceptance up to the present time.

Our knowledge of the signs revealed by auscultation has received certain not unimportant additions *since the time of Lænnec;* amongst these should be mentioned the sound of pleuritic friction (Reynaud, 1829), that of pericardial friction (Collin, 1831), and the proper interpretation of the sounds heard over the vessels, and of many other facts connected with the auscultation of the heart and abdomen. (See the chapters in which these points are discussed.)

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[*] . . . . . . *ἕτερος μὲν τὰς χεῖρας ἐχέτω. σὺ δὲ τὸν ὦμον σείων ἀκοάζεσθαι, ἕως ὁκότερον ἂν τῶν πλευρέων τὸ πάθος ψοφέῃ.* (Another holds the hands of the patient, whilst thou, shaking him by the shoulders, listenest from which side the sound proceeds.)

[†] . . . . . *τρίζει τὸ πνεῦμα οἷον μάσθλης* . . . . . (the respiratory sound is attended by a creaking, as of leather.)

Another passage, relative to the presence of fluid in the chest, is to the following effect:—*τούτω ἂν γνοίης, ὅτι οὐ πῦον, ἀλλὰ ὕδωρ ἐστὶ, καὶ ἢν πολλὸν χρόνον προσέχων τὸ οὖς ἀκουάζῃ πρὸς τὰ πλευρὰ, ἐστὶν ἔσωθεν οἷον ψόφος.* (Hereby mayst thou know that the fluid is water, not pus,—by holding thine ear to the side and listening, when it is as if a sound came from within.)

Catarrhal sounds also are repeatedly mentioned by Hippocrates. A very complete collection of the passages in which he treats of the above subjects and of physical diagnosis generally, has been made and published by Küchenmeister.

METHODS OF AUSCULTATION.

Of these there are two, *immediate* and *mediate;* in the former the ear is applied directly to the chest, in the latter the stethoscope is interposed.

1. Immediate auscultation has these advantages, that the sounds generated within the respiratory apparatus are heard more loudly with the unaided ear than through the stethoscope, and that a larger area can be examined at a time; when it is necessary, therefore, that the chest should be gone over rapidly, especially its posterior surface, (as in very exhausting diseases, in which the patient is unable to sit up any length of time), immediate auscultation is to be preferred.

It has many drawbacks, however; not only is it, from the form of the thorax, attended by great inconvenience both to the patient and to the examiner, but it also involves many sources of error. It is frequently a matter of considerable difficulty, sometimes it is even impossible, to apply the ear accurately to certain parts the exploration of which is of the utmost importance,—the supraclavicular regions, for instance, especially when, as is so commonly the case, they are hollowed out by emaciation. In the supraspinous regions also this method is usually equally inapplicable.

The practice of immediate auscultation is, further, always associated with certain circumstances which are very apt to mislead; thus, the rubbing of the hair in the neighbourhood of the ear on the surface of the thorax in inspiration, and to a certain extent also in expiration, may give rise to artificial sounds which, as they closely simulate the so-called crepitant râles, sometimes prove very deceptive. And in those cases in which abnormal sounds are strictly limited in distribution, or in which the sounds vary greatly in character within a small area, immediate auscultation is obviously an untrustworthy method of examination.

Auscultation is best practised on the exposed skin. When female delicacy absolutely forbids this,—which comparatively seldom happens, most of the laity being fully aware of the great importance of auscultation,—the parts may remain covered by the chemise; and although in such circumstances perplexing friction-sounds are usually mingled with the proper respiratory murmur, the practised auscultator soon learns to recognise these and to distinguish them from the sounds really originating in the organs of respiration. A skilful examiner is able also to appreciate, through the clothes, both the normal and the abnormal respiratory sounds. No one will, of course, rest completely satisfied with such a superficial examination, more particularly when the disease is in its earlier stages and when it is desired to determine exactly its extent and nature.

2. Mediate auscultation, by means of the stethoscope. The various forms of stethoscope, though there are a great many in general use, are widely known and need little description. In the most useful form of instrument the ear-plate is somewhat concave and fits closely to the external ear, so as to facilitate the free and unobstructed entrance of the waves of sound. This condition is still more completely fulfilled when the stethoscope is furnished, not with an ear-plate, but with a small conical plug which is introduced into the auditory meatus; the flexible stethoscope, the auricular and thoracic ends of which are connected by a tube of india-rubber, is constructed in this fashion.—All the acoustic phenomena are heard much more distinctly through the binaural stethoscope,—a simplification of that so much used in America, having one cup-shaped thoracic end from which proceed two flexible tubes, each provided with a small conical ear-piece to fit the meatus. But its great advantage, the exceeding clearness with which it conveys sound, is nullified by an objection which even long practice is not always able to overcome, namely, that the slightest movement of the tube or of the ear-piece in the ear excites loud accessory sounds.—This variety of stethoscope is obviously well suited for self-auscultation.

In applying the ear to the stethoscope it is necessary to guard against pressing too firmly on the thorax, as this is decidedly unpleasant even to persons in good health, and is often positively painful to those suffering from any illness, especially if they are to any extent emaciated. In cases in which the acoustic phenomena are feeble and difficult to catch, the ear which is not employed in auscultating may be filled with cotton-wool, to exclude such sounds as would be likely to confuse those coming from within; a little experience, however, renders this unnecessary.—In auscultating the lungs the examination should be begun at the apices and continued regularly downwards towards the bases, the symmetrical spots on each side being compared with each other, though this latter part of the proceeding is not absolutely indispensable.—The sounds may be rendered more audible by causing the patient to breathe deeply, especially if the respiration be shallow.

The objects aimed at in auscultating the lungs are :

I. To obtain a knowledge of the sounds which accompany both of the respiratory acts. These fall naturally into three groups ;
   a. *Simple respiratory murmurs ;*
   b. *Râles*, produced during respiration, by the presence of fluid in the bronchi or in the substance of the lung ;
   c. *Friction sounds*, arising from the rubbing of the roughened pleural surfaces on each other.

II. The auscultation of the cough and voice.

SIMPLE RESPIRATORY MURMURS.

Strictly speaking this expression includes only those breath-sounds which may be heard over the *thorax ;* more generally, it comprehends also those which originate, during respiration, in the nose, mouth, and larynx. The sounds produced by the current of air in the nasal and buccal cavities (the mouth being open) and in the pharynx not unfrequently, especially if the breathing be hurried or irregular, become intermingled with and obscure the true respiratory phenomena, and as they possess the same blowing character as the laryngeal and bronchial sounds they often prove a source of great confusion to beginners. To eliminate the buccal sound the patient should be made to close the mouth and to breathe only through the nose ; with regard to the nasal and pharyngeal sounds it is necessary to be able to recognise them readily and to distinguish them from the pulmonary respiratory murmur, as they are sometimes audible even over the whole of the thorax. The laryngeal sounds do not complicate the auscultation of the chest, as, if the laryngeal mucous membrane be normal and the passage of the current of air be unimpeded, they are quite inaudible over the thorax, except at certain parts, which are mentioned in detail on p. 130.

The simple respiratory sounds produced in the lungs, both in health and in pathological conditions, are divided by Skoda into three groups:

1. *Vesicular respiratory murmur.*
2. *Bronchial respiratory murmur.*
3. *Indeterminate respiratory murmur.*

THE VESICULAR RESPIRATORY MURMUR.

The characteristic features of this sound may be easily reproduced by nearly closing the lips and gently drawing a current of air inwards; there is obtained in this way a sighing or slightly whiffing sound, which closely resembles that of vesicular respiration.—The vesicular respiratory murmur owes its name to the circumstance that *it is produced at the instant at which the air enters the alveoli.* That it is generated just at this stage of the respiratory act seems placed beyond doubt by the fact that the sound disappears in all cases in which the entrance of air into the vesicles is prevented (as when they are filled with fluid), even though the bronchi, from the largest to the smallest, be perfectly free and pervious.—With respect to the actual cause of the vesicular murmur no adequate explanation can be given. Whilst most hold that it is no way connected with the bronchial sound, some authors have lately attempted to show that it is merely a modification of the sound originating in the larynx,—that the sonorous waves, in passing from that part into the relatively wide resonant chamber presented by the lungs, travel more slowly, are reduced in force and intensity, and lose the timbre and other properties they possessed while still in the larynx (Baas). It is undoubtedly the case that the laryngeal sound suffers considerable alteration in transmission through permeable, air-containing lung tissue. If, for example, a piece of the lung of some animal be inflated (but not too much) and laid on the larynx the characteristic laryngeal sound is no longer heard through it, but a sound which, in proportion to the thickness of the portion of lung used, is less tubular in quality and less clear in tone, and which may even possess all the properties of the ordinary normal vesicular murmur (Penzoldt); but if a piece of *liver* be substituted for lung the laryngeal sound is heard through it quite unchanged. Possibly the sound of vesicular respiration noticed in the experiment described may have had its origin not merely in the propagation of the laryngeal accessible to the air, and are rather later in expanding, than the freely permeable lung-substance lying between them, and inspiration becomes jerking or interrupted. After repeated deep inspiration, or an attack of coughing, it disappears for a time, but returns shortly afterwards.—From the physical point of view interrupted inspiration indicates merely the existence of some obstacle to the entrance of air into the pulmonary parenchyma; this obstacle is sometimes of short duration, and then is of no particular moment; at other times it is found to persist for a considerable period, and then may be accepted as pointing to incipient catarrh in the apices. In such cases also there are usually other auscultatory phenomena (prolonged expiration, perhaps even a few feeble râles) which warrant the same diagnostic inference.

Another modification of the normal respiratory sound is the so-called *systolic* vesicular murmur, sometimes heard at the borders of the lungs, near the heart; it is caused by the sudden rush of air into the alveoli of these parts of the lung, which rapidly expand and occupy the space rendered vacant by the shrinking of the heart in systole. This sound, nevertheless, occasionally occurs quite independently of inspiration, and in such circumstances is very feeble, though most commonly it is perceptible only when inspiration happens to coincide with a cardiac contraction,—when the portions of lung immediately adjoining the heart receive more air than the other parts of the lung.

The only diagnostic conclusion that can be drawn from the existence of the vesicular respiratory sound is that wherever it is audible the subjacent lung-tissue is permeable to air; but it does not necessarily follow that the part is capable of admitting a *normal quantity of air*.—The expansibility of the lung may, from various causes, be considerably diminished without involving any marked alteration in the vesicular murmur, so long as between these denser parts others intervene still having the normal spongy texture; and any deficiency is still further masked by the breath-sounds proceeding from the adjoining healthy tissue. In all these cases, however, the diagnostic inference, that the part under examination has suffered *diminution* of its air-contents, is rendered easier by the presence of other physical signs, such as changes in the percussion-note sound through other resonant spaces, but also in the simultaneous vibration of the inflated and tense lung-substance (see below).

The various theories regarding the cause of the vesicular respiratory murmur are the following:—

According to Læннec, Skoda, and others, it arises from the friction of the air against the walls of the alveoli while the latter are being dilated; the results of experimental investigation, however, tell against this view, as the inflation even of large chambers (large caoutchouc balloons, for instance) is accomplished noiselessly. If the ultimate ramifications of the bronchi be represented by a piece of indian cane (which consists of extremely fine tubules of equal diameter), and to this a thin elastic bladder be attached, it is found that on blowing through the cane and distending the bladder no sound is produced.—Others ascribe the vesicular murmur to vibration of the air as it enters the alveoli; the condition necessary to the occurrence of this "oscillation" of the current of air Chauveau, Bondet, P. Niemeyer, &c., professed to find in a constriction of the most minute bronchioles which takes place at the point at which they open into the funnel-shaped infundibula; they thus held that the vesicular murmur is made up of numberless stenosis-murmurs.—Talma adduces certain experiments, in which a current of air was forced through india-rubber tubes, in support of the hypothesis that the vesicular murmur is caused by the "friction of small bodies of air on each other." Over the middle of these tubes the bruit heard is continuous, while on contracting the orifice of entrance the sound becomes weaker at the middle of the tubes, but stronger at the constricted point; from these facts he infers that the sound heard at the centre originates at that part (from friction of the particles of air on each other) and is not transmitted to it from the orifice.—Baas, in opposition to those who look upon the vesicular murmur as an independent phenomenon, unconnected with the other respiratory sounds, holds that the tracheal sound is the one source of all the pulmonary respiratory murmurs, and that it is merely variously modified at different parts according to the dimensions of the resonant spaces to which it is conducted; thus, in the bronchi it becomes bronchial, in the alveoli vesicular.—Gerhardt and Penzoldt attribute the vesicular murmur to still another cause, to vibration of the lung tissue, as its state of tension is increased during inspiration; they suppose that these vibrations, when added to the bronchial sound, give it the vesicular character. Penzoldt was led to that conclusion by the following experiment: if a stethoscope, formed of several separate parts screwed together, having a thin tense membrane (the air-bladder of a fish) interposed between each two pieces to represent the tissue of the lung when distended, be applied over the larynx, or over those parts of a diseased lung at which, with an ordinary instrument, the respiration is bronchial, the sound now heard is no longer bronchial but vesicular.

The vesicular respiratory murmur is audible *only during* *inspiration*, usually throughout its whole duration, but only towards the end of the act if breathing be superficial. The intensity (clearness) of the sound depends in most cases on the energy with which respiration is carried on; in general, however, it varies greatly in different individuals, even though their lungs be equally well developed and respiration be equally energetic. These differences have no great diagnostic significance.

In health the vesicular murmur is heard over the whole thorax, most loudly at those parts where the soft coverings are thinnest and offer least opposition to the propagation of the sound. The inspiratory murmur is therefore louder anteriorly than posteriorly, loudest in the infraclavicular region, feeblest in the supra- and infra-spinous regions; it is also weak wherever the subjacent layer of lung is thin,—at the apices and the anterior and inferior borders. The two sides of the chest often show marked differences in the intensity of the sound, the latter being louder sometimes on one side, sometimes on the other. Occasionally, though not always, there are obvious local causes for the enfeeblement of the murmur at certain parts,—such as increased thickness of the soft parts and abnormal convexity of the ribs, both of these conditions being highly unfavourable to the transmission of sounds.—If inspiration be sufficiently vigorous the vesicular murmur may be perceived not only in those parts behind which lung substance is situated, but also at other parts —the hepatic region, for instance,—to which the sound is conducted.

With regard to the character of the vesicular respiration, a distinction is made between the *soft* and the *harsh* or *rough* vesicular murmur. In normal circumstances, when the mucous membrane lining the air-passages is in a healthy state, the sound is soft; but in *catarrh and swelling of the membrane*, it becomes *rough*, the normal sound being altered by the addition of vibrations from the folds and prominences on the mucous surface. Following the distribution of the catarrhal affection, this harsh respiratory murmur may be strictly circumscribed or may be diffused over the whole thorax.—The detection of rough respiration over a limited area is of diagnostic importance as serving to determine the cause of the catarrh. Experience shows, for example, that catarrh of the apices is seldom primary, but usually secondary, indicating the beginning of caseous condensation in these parts. A rough respiratory murmur, therefore, localised in one or both apices, presenting no variation as regards its site on repeated examination, furnishes good ground for the suspicion that the catarrh is secondary to a phthisical affection; the same sound, however, is heard over the whole of one or both lungs, both in primary simple bronchial catarrh and in the secondary catarrh associated with various diseases of the lung-substance and air-passages.

The harsh vesicular murmur occurs sometimes alone, but more frequently combined with certain accessory sounds,—the râles, to be considered further on.

Harsh respiration is, nevertheless, quite consistent with perfect health; thus, in children, till they are about twelve years of age, the vesicular murmur is exaggerated and rough, and so characteristic is this sound that harsh respiration generally is often spoken of as *puerile*. This peculiarity of the respiratory murmur in children may be due to the fact that comparatively little opposition is offered to the transmission of sound by the thin chest-wall, and to the resistance to inspiratory expansion presented by the greater elasticity of the lungs in early life.—After the twelfth year the peurile breath-sound passes gradually into the softer vesicular murmur of adults.

A special variety of the vesicular respiratory bruit is the *jerking* inspiration. This term is used to describe a vesicular inspiration divided into two or more parts,—a phenomenon which may be imitated by contracting the lips and drawing the air through them in several quick, sudden draughts repeated at short intervals. Such an interrupted inspiratory sound is often audible over a large extent of the thorax when the individual under examination breathes irregularly or very slowly, in such a way that the air gains earlier access to one part of the lung than to another. A quick deep inspiration, by which the lungs are speedily and evenly expanded, causes this physiological variety of jerking respiration to disappear.—From this is to be distinguished an interrupted vesicular murmur of pathological origin, occurring in one or both apices, and usually confined to these parts. Thus if the air-cells in the apices be partially occupied by infiltration, and the finer bronchi be also somewhat reduced in calibre by tumefaction of their mucous membrane, the affected portions of the lungs are less readily accessible to the air, and are rather later in expanding, than the freely permeable lung-substance lying between them, and inspiration becomes jerking or interrupted. After repeated deep inspiration, or an attack of coughing, it disappears for a time, but returns shortly afterwards.—From the physical point of view interrupted inspiration indicates merely the existence of some obstacle to the entrance of air into the pulmonary parenchyma; this obstacle is sometimes of short duration, and then is of no particular moment; at other times it is found to persist for a considerable period, and then may be accepted as pointing to incipient catarrh in the apices. In such cases also there are usually other auscultatory phenomena (prolonged expiration, perhaps even a few feeble râles) which warrant the same diagnostic inference.

Another modification of the normal respiratory sound is the so-called *systolic* vesicular murmur, sometimes heard at the borders of the lungs, near the heart; it is caused by the sudden rush of air into the alveoli of these parts of the lung, which rapidly expand and occupy the space rendered vacant by the shrinking of the heart in systole. This sound, nevertheless, occasionally occurs quite independently of inspiration, and in such circumstances is very feeble, though most commonly it is perceptible only when inspiration happens to coincide with a cardiac contraction,—when the portions of lung immediately adjoining the heart receive more air than the other parts of the lung.

The only diagnostic conclusion that can be drawn from the existence of the vesicular respiratory sound is that wherever it is audible the subjacent lung-tissue is permeable to air; but it does not necessarily follow that the part is capable of admitting a *normal quantity of air*.—The expansibility of the lung may, from various causes, be considerably diminished without involving any marked alteration in the vesicular murmur, so long as between these denser parts others intervene still having the normal spongy texture; and any deficiency is still further masked by the breath-sounds proceeding from the adjoining healthy tissue. In all these cases, however, the diagnostic inference, that the part under examination has suffered *diminution* of its air-contents, is rendered easier by the presence of other physical signs, such as changes in the percussion-note (muffling of the sound) and certain auscultatory phenomena, especially fine bubbling râles.

If the air be *entirely excluded* from a larger or smaller portion of the lung the vesicular murmur within the region so affected is lost, being either totally suppressed, or replaced by a sound of no determinate quality or by bronchial breathing.

The vesicular respiratory murmur is *abolished* over a large area, sometimes even over the whole of one side of the chest, when the lung is *completely collapsed* by the *pressure of fluid* or *air* in the *pleural cavity* or by any other cause; it disappears to a less extent when the lung is compressed by mediastinal tumours, large pericardial effusion, an excessively hypertrophied heart, &c.; it becomes inaudible on both sides in very aggravated cases of vesicular emphysema; and finally, when the lung is completely *solidified* the vesicular murmur is no longer heard, but is replaced by other auscultatory signs.

The vesicular respiratory murmur is *weakened*, over a greater or less extent of surface, by the presence of minor degrees of the above-named affections, by marked reduction in the calibre of the larynx and trachea; temporarily, also, by obstruction of any of the larger bronchi.

Such constrictions of the air-passages may result from the formation of croupous membranes, swellings, cicatricial (syphilitic) adhesions, &c., more rarely from paralysis of the dilators of the glottis on both sides; when it arises from the latter cause the rima glottidis, instead of dilating in inspiration, contracts, sometimes even to absolute closure, the vocal cords falling asunder again only in expiration.—In cases of obstruction of the bronchi by superabundant mucous secretion the vesicular murmur is weakened only in circumscribed spots, and regains its intensity after an attack of coughing. If the embarrassment of respiration be due to the partial or *complete closure* of one of the *principal bronchi* by the impaction of a foreign body (a somewhat rare occurrence), the vesicular murmur over the greater part of one side of the chest is so enfeebled as to be almost inappreciable.

Weakening, or even abolition, of the respiratory sounds may thus spring from so many diverse causes that it admits only of the general diagnostic interpretation,—that the air gains access to the alveoli with difficulty or not at all.

In those pathological conditions in which vesicular respiration is abolished (in cases of compression of the lung by pleuritic exudation or air, or of severe pulmonary emphysema, for instance), inspiration is attended either by no audible sound or only a faint, undefined whiffing generated in the bronchi.—In another group of cases, characterised by *complete infiltration* of the air-cells, the respiratory sound is no longer vesicular, but bronchial, and accompanied usually by râles, when the bronchus leading to the hepatized part is not stopped-up by secretion. The vesicular murmur, in becoming weaker, becomes also less clearly defined in character, till it passes gradually into the indeterminate respiratory murmur.

THE EXPIRATORY MURMUR.

In auscultating the normal chest in expiration only a weak and soft *whiffing* or buzzing, *indefinite* sound is heard, which bears no trace of resemblance to the vesicular inspiratory murmur; it is usually much shorter than the latter, and is produced by the outward current of air through the bronchi.

The principal pathological alterations of the expiratory murmur are *prolongation* and *harshness*. Very frequently, indeed usually, both changes are observed together and are due to the same cause.

Prolongation of the expiratory murmur occurs over extensive or circumscribed areas, the breath-sounds being either otherwise unaltered or accompanied by accessory sounds. It always indicates that there exists some hindrance to the free escape of the respired air. It is one of the physical signs, therefore, that attend severe bronchial catarrh, being caused by the diminution in the lumen of the bronchi which takes place from swelling of their mucous lining; it is met with particularly in the diffuse bronchial catarrh which is associated with vesicular emphysema.—It is also very frequently heard, but more strictly localised, as the result of the bronchial catarrh connected with condensation of the lungs. Limited to one or both apices, prolonged expiration is one of the earliest signs of incipient catarrh, and may then be regarded as evidence of the commencement of cheesy degeneration; in such circumstances it is often combined with other auscultatory phenomena (dry or moist râles) pointing to the same conclusion.

A *harsh* expiratory murmur, like one which is prolonged, being caused by obstruction to the expiratory current of air and vibration of the swollen bronchial mucous membrane, indicates equally clearly the presence of bronchial catarrh. It merges gradually into that form of expiration to which the accessory sounds,—particularly the dry râles, sonorous, buzzing, sibilant,—are added; these râles, however, may be so loud as to completely cover the proper expiratory sound.

THE BRONCHIAL (LARYNGEAL, TRACHEAL) RESPIRATORY MURMUR.

In auscultating the larynx there is heard, both in inspiration and in expiration, a loud, rough murmur, which may best be compared with the sound of the aspirate "h," or with the puffing sound produced by blowing through a tube, such as that of the stethoscope. The pronunciation of the German word "hauchend" reproduces fairly the peculiar characters of the laryngeal respiratory sound; when the latter is soft the quality of the "h" predominates, when rough, that of the guttural "ch." The laryngeal sound is also sometimes described as *tubular*, from its complete identity with the hollow, blowing sound caused by the rushing of air through a tube. It is loudest at the rima glottidis, this being the narrowest part of the tube through which the aerial current passes. From the larynx it is propagated downwards into the trachea and both bronchi, but with gradually decreasing intensity; in the trachea, however, it is quite as loud as in the larynx, and is of almost equal strength in inspiration and in expiration. Opposite the bifurcation of the trachea, (between the shoulder-blades and near the fourth dorsal vertebra), the laryngeal respiratory murmur is already considerably feebler and softer, and is clearly audible only in expiration; it is more distinctly appreciable on the right side than on the left, the right bronchus being wider than the left and lying nearer to the surface of the thorax. From the point at which the trachea divides the tracheal murmur is carried onwards into the bronchi, so that it may sometimes be heard over the whole interscapular, and occasionally even in the supraspinous, regions.

Lippe examined 203 healthy individuals, to determine the limits of the area of bronchial respiration. He found that it was invariably bounded superiorly by the seventh cervical vertebra, that it extended downwards from that point and to both sides of the chest, in only four cases being limited to the right side. Inferiorly its boundary varied, being usually marked by the second, fourth, or sixth cervical vertebra, and but rarely reaching further downwards.

Outside of the region described bronchial respiration is not heard in the normal thorax, although, as may be inferred from certain pathological phenomena, the laryngeal sound is conveyed even to bronchi of medium calibre, its tubular quality being obviously modified or suppressed by the low conducting power of the spongy lung-tissue.

In *pathological* conditions bronchial respiration may become audible at any part of the thorax, and during both inspiration and expiration; it is usually louder during expiration, rarely louder during inspiration.

The pathological bronchial respiratory murmur is, in well-marked cases, very similar in character to the laryngeal sound, that is, it has almost the same pitch and timbre, so far as it can be said to possess these properties of a true musical tone; but in the majority of cases it resembles more nearly the bruit heard at the bifurcation of the trachea, which is softer and weaker than the laryngeal sound, and which, therefore, has a better claim to the attention and study of beginners, to whom the correct appreciation of the qualities of bronchial breathing always presents considerable difficulty, as the type of bronchial respiration.—It frequently happens that the bronchial character of the sound is not so pronounced as usual, and in such circumstances it is easily mistaken for harsh and prolonged expiration; no sure guide can be given for the accurate discrimination of these phenomena, except the already-mentioned aspirate quality of "h" or the guttural quality of "ch" in the bronchial sound. Practice and experience can alone confer the power of forming a certain diagnosis when the sounds are badly defined, or when the bruit is of the indeterminate class, on the border-land between bronchial and vesicular respiration. These indistinct respiratory murmurs are designated as "somewhat bronchial", "obscurely bronchial", or "approximately bronchial" sounds.

In bronchial respiration of pathological origin two qualities are distinguishable,—it may be *harsh* or *soft*, *high-pitched* or *low-pitched*. It becomes rough when the tracheal or bronchial mucous membrane is swollen, and the lumen of the bronchi in that way diminished. Similarly the bronchial murmur may take on an excessively harsh character, audible even at some distance from the patient, when the upper part of the wind-pipe is compressed by glandular tumours or reduced in calibre from other causes,—croup in children, diphtheritis of the larynx and trachea, &c. It is particularly in these cases that the tracheal bruit is conducted to any great distance downwards into the bronchi, when in the whole of the upper part of the thorax the vesicular murmur may be accompanied by the sound generated in the trachea.—The circumstances which give rise to variation in the pitch are less clearly understood; these differences, however, have no diagnostic importance, as in the same patient there are often rapid alternations of high-pitched with low-pitched bronchial breathing without any change in the other physical signs of the disease.—The *intensity* of bronchial respiration is also very variable; the sound is sometimes almost as loud as at the level of the larynx or trachea, at other times it is very feeble, as if it proceeded from some distant part. These differences are due, apart from the energy with which respiration is carried on, more particularly to the nature of the morbid process in which the bronchial breathing has its origin, and to certain other factors still to be discussed.

THE CONDITIONS WHICH GIVE RISE TO BRONCHIAL RESPIRATION.

Bronchial respiration occurs in cases of *pulmonary cavity* and of *condensation of the lung-tissue*, under the following conditions:—

1. In *pulmonary caverns*, when these are situated superficially, surrounded by rigid and dense walls, and at least so large as to involve one of the larger bronchi, the air of which freely communicates with that of the cavity on the one hand and with that of the trachea on the other. In the absence of any of these conditions bronchial respiration ceases, or loses its distinctive character: thus, if the excavation be deep-seated, covered over by normal air-containing lung-substance, the bronchial quality of the respiration in the cavity is lost in the vesicular sound coming from the expansible tissue; even if the vomica be near the surface, but invested by healthy lung, the waves of sound of bronchial quality from the former are scattered and broken up by the adjoining lung-substance, which is of feeble conducting power. Further, if the cavern be too small to be in free communication with a bronchus of moderately large calibre, the sonorous undulations are propagated from the larynx so feebly as to be inaudible. And finally, should the bronchus leading to the cavity be occluded by mucus the production of the bronchial sound is obviously rendered impossible, as the respiratory current cannot

penetrate beyond the obstruction; but as soon as free intercommunication is re-established by coughing and expectoration, the bronchial bruit reappears. Its intensity, in cases of pulmonary excavation, depends, other things being equal, on the size of the cavity, on the diameter of the bronchus opening into it, and on the energy of the act of respiration.

2. Bronchial respiration is observed in partial or complete *condensation* of the lungs, whether the result of compression or infiltration of the air-cells.

The highest degree of infiltration takes place in pneumonic hepatization; here, therefore, bronchial respiration is louder than in cases of less complete consolidation from other causes. That the hepatized portion of lung may present a sound of bronchial quality it must fulfil the same physical conditions that have been already described as necessary in the case of pulmonary cavities. The larger the part hepatized, and the greater, therefore, the number of bronchi ramifying in it, the louder the bronchial sound. As pneumonia attacks most frequently the lower lobes bronchial respiration is most common in the posterior lower regions of the chest; and when the whole of the lower lobe is affected the bruit is audible from the base of the lung up to the middle of the shoulder-blade.—The bronchial respiratory murmur in pneumonia disappears at the beginning of the stage of resolution, when the air again gains access to the alveoli.

In cases of condensation of the lungs from other causes, such as caseous degeneration, chronic interstitial pneumonia with consecutive dilatation of the bronchi, &c., the parts so affected frequently have isolated patches of healthy tissue scattered in their interior; bronchial respiration in these circumstances is much less intense than in the hepatization of pneumonia. More often bronchial respiration is obscured by the presence of râles originating in the air-passages; after coughing, however, especially if it result in free expectoration, the bronchial character of the respiratory sound comes out more distinctly. But if the portions of spongy texture, enclosed within the indurated tissue in the above instances, be relatively of large size the respiratory murmur may be almost perfectly vesicular. In all varieties of condensation of the lungs, from whatever cause arising, the same conditions are necessary to the production of bronchial respiration as in pneumonic hepatization, which simply serves as the type of the most complete solidification.

Bronchial respiration may also be observed in all those conditions in which *the lung is rendered void of air by compression.* The most marked degree of pulmonary collapse takes place from pleuritic exudation, that from pneumothorax being scarcely less complete; more partial collapse is caused by copious pericardial effusion, large aneurisms of the aorta, excessive hypertrophy of the heart, morbid growths within the pleural sac, and tumours of the abdominal organs, which push the diaphragm upwards. Nevertheless, bronchial respiration occurs in but a small proportion of the cases of pleuritic effusion, and only when the fluid is moderately abundant, that is, when the air-cells and finer air-tubes, not the larger bronchi, are pressed upon; *small* effusions occasion no bronchial respiratory murmur, as they do not usually exercise sufficient pressure to drive all the air from the lungs, but produce only retraction of these organs, reducing somewhat the quantity of air they contain. The weight of a great mass of fluid in the pleura, on the other hand, tends to obliterate also the larger bronchi, and so to hinder the free transmission of the waves of sound from the larynx downwards into the lungs. Further, on the front of the chest, in cases of pleuritic exudation, bronchial respiration is never heard, as the fluid comes between the lung and the anterior wall of the thorax, and to a great extent absorbs or suppresses the respiratory murmur. But posteriorly, where the shrunken and dense lung is in close contact with the chest-wall, the bronchial sound transmitted from the trachea is heard in considerable intensity.—It is a much rarer event to find the lung compressed to such a degree as to favour the production of bronchial respiration, from the presence of air in the pleural cavity than from pleuritic effusion; in such cases there is generally no respiratory murmur, or only one of indeterminate character, which is sometimes accompanied by a metallic echo (see "amphoric respiratory murmur," p. 139).

The other morbid changes above enumerated, giving rise to compression of the lungs, very seldom induce a state of complete collapse in the parts concerned, so that the respiratory sound does not commonly, from these causes, assume the unmistakeably bronchial quality.

PHYSICAL CAUSE OF PATHOLOGICAL BRONCHIAL RESPIRATION.

The bronchial respiratory murmur heard in disease is nothing more than the laryngeal sound, carried with scarcely diminished intensity through consolidated lung-tissue. This was the explanation adopted by Lænnec, though in certain details his views must be held as erroneous. His theory is to the following effect:—in normal conditions the bronchial sound is so blended with the vesicular sound as to be inaudible as an independent phenomenon; but if the pulmonary tissue become consolidated, and the vesicular respiratory murmur in that way abolished, the bronchial bruit is heard, not merely in its original intensity, but more strongly, as the *dense, airless lung-substance is a better conductor of sound.*

This explanation is inaccurate in only one point. The occurrence of bronchial respiration is not dependent merely on the disappearance of the vesicular murmur, for if that were the case respiration should be distinctly bronchial in severe pulmonary emphysema, in which every trace of the vesicular sound is lost. But the second part of Lænnec's proposition—that consolidated lung-tissue is a good conductor of sound, and that thereby the bronchial bruit is rendered audible,—must be accepted as well-founded, notwithstanding the objections urged against it by Skoda (see p. 136).

It is a matter of daily observation that the cardiac and vascular sounds are conveyed to the thoracic parietes in a much more intense form through indurated than through expansible lung; when condensation takes place in the left apex the sounds from the pulmonary artery, when in the right those from the subclavian, are heard with great distinctness. Similarly, marked increase in the size of the liver, or the presence of morbid growths in its immediate neighbourhood, magnifies the sound of the abdominal aorta. The reason that dense pulmonary tissue forms a better conductor of sound than that which is normal and expansible, is that the structure of the former is homogeneous, whilst that of the latter is not, the air contained in the alveoli alternating with the bands of tissue constituting the parenchyma of the organ; and when a sound is made to traverse media differing in consistence it is weakened and finally suppressed.

These observations show that it is necessary to modify Læennec's theory, and somewhat in the following manner. In health bronchial respiration is heard only at the larynx and over the trachea and its bifurcation, which lies close to the chest-wall; but from the point at which the bronchi enter the lung bronchial respiration disappears, as, though sounds of bronchial quality are undoubtedly transmitted to the larger, perhaps also to the medium-sized air-tubes, they never reach the surface, being arrested in their course by the pulmonary tissue, which, being non-homogeneous, is a bad conductor. But should the lung become consolidated, and thereby acquire the property of conducting sound clearly, the bronchial murmur is conveyed with undiminished intensity to the thoracic parietes, provided that the portion of lung affected is of sufficient size to include a large bronchus, in which tracheal respiration is audible.

Skoda has sought to overthrow Læennec's teaching, which ascribes bronchial respiration to the superior conducting-power of a condensed lung. His objections are based on a long series of experiments, the general tenour of which led him to the opposite conclusion,—that as a conductor of sound expansible, air-containing lung-substance far excels consolidated tissue. One of the simplest of these experiments may be here mentioned: two lungs, one normal, the other hepatized, are removed from the thorax and examined by applying a stethoscope to its surface; into this instrument one experimenter speaks, while another auscultates the voice through another stethoscope. As the result of this comparative investigation it was found that the sound was heard over a somewhat more extensive area in the healthy than in the hepatized lung.

Choynowski obtained very different results. In his experiments he eliminated one likely source of error,—the varying intensity of the human voice, and substituted for it the constant sound given by an ordinary watch; this was, further, held as far as possible from the auscultating ear and surrounded by substances of low conducting power, to exclude the possibility of the transmission of the ticking sound through the air. The result was that the sound was carried with *greater intensity* through *hepatized* than through normal lung.—Bondet and Chauveau, also, having introduced a cannula into the trachea of a horse suffering from pneumonia, and whispered into the instrument, heard the voice clearly over the hepatized lung, but not over the healthy lung.

Skoda asserts further that to the production of a bronchial respiratory murmur as loud as that which is usually audible over hepatized tissue a strong current of air is necessary; such a current, however, can scarcely exist in the hepatized lung, as it neither expands in inspiration nor contracts in expiration, its volume remaining unchanged throughout the whole of the act of respiration. In opposition to this Choynowski justly observes that the air in the bronchi *is* in motion, even when the alveoli are completely infiltrated; its density varies in respiration, becoming less in inspiration and greater in expiration, provided that it is in free communication with the column of air in the trachea. But when the air contained in two communicating cavities is subjected to unequal pressure, a current is at once set up from the chamber in which the density is greatest to that in which it is least; and this takes place in the lung during each new inspiration. The probability of the existence of such a current is also rendered more evident by the fact that in a completely consolidated lung very minute râles are frequently heard, which obviously can originate only in the finest ramifications of the bronchi.

Skoda combats Lænnec's views on the further ground that in hepatized tissue bronchial respiration is instantaneously abolished when the bronchus is closed by a plug of mucus, and returns equally quickly when the obstructing body is expelled and free communication re-established. If condensed tissue, argues Skoda, were a better conductor of sound than normal lung, it should make no difference whether the bronchi of the portion of lung concerned be pervious or impervious. —But conduction has nothing to do with this phenomenon, as the disappearance of the respiratory murmur on occlusion of the bronchus is caused simply by the inability of the waves of sound to penetrate the hepatized tissue on account of the opposition offered by the obstruction.

In the same way may be refuted Skoda's objection, that if dense bodies are really the best conductors of sound the respiratory murmur and voice should be louder, not weaker, in cases of pleuritic exudation. It is the fact that very frequently bronchial respiration is heard in the interscapular space, close to the vertebral column, where the compressed and airless lung is closely applied to the thoracic wall; but on the front of the chest no respiratory sound is perceptible, as the lung is separated from it by the mass of fluid exudation; the sonorous vibrations, therefore, in their passage through non-homogeneous media, lung and fluid, are so considerably enfeebled as to be no longer audible on reaching the surface or the auscultating ear.

Skoda has endeavoured to define bronchial respiration as a *phenomenon of consonance.*

The basis of this consonance-theory, which will be described in detail on another page (see "Bronchophony"), is that bronchial respiration is generated or magnified in caverns and in the bronchi of condensed lung substance by the air in these cavities and bronchi vibrating in consonance with that of the trachea; the condition necessary for this consonance is provided in the circumstance that the air is pent up in confined spaces, whose *solid* walls (the indurated parenchyma surrounding the bronchi) reflect the sonorous undulations.

This theory is capable of but a very limited application, having a bearing only on those cases in which the bronchial respiration is of metallic character, or is accompanied by an amphoric echo (in caverns of large dimensions) or by râles of metallic timbre. But here *it is not the bronchial respiration, but the metallic (amphoric) quality* which is added to it and to the râles, that constitutes the *consonance-phenomenon.* —Skoda bases his theory almost exclusively on the ground that in it may be found the explanation of his observation that bronchial respiration is sometimes *louder* over the thorax than over the trachea. Such cases have never yet come under my observation; at best they must be exceedingly rare.

The Metamorphosing Respiratory Murmur.

(Das Metamorphosirende Athmungsgeräusch.)

This is a special modification of bronchial respiration. It has, according to Seitz, who was the first to describe it, the following characters: it occurs only in *inspiration;* at the outset it is distinguished by an unusual harshness, which is quite different from that of harsh vesicular breathing, and which may be imitated by placing the tongue against the palate, as in pronouncing the letter g (hard), and making a forcible inspiration; the sound so obtained is that which is so often observed in *stenosis* of the bronchi, as in the diffuse bronchial catarrh associated with emphysema of old standing. This harshness lasts only about one-third of the inspiratory period, when it suddenly ceases, and gives place, during the rest of the inspiration, to bronchial breathing accompanied by a metallic echo, or to ordinary râles.

Seitz has met with this variety of respiration only in cases of cavity in the lungs (most often in the upper lobes), and has accordingly classed it as a cavernous phenomenon.—The harsh sound with which metamorphosing respiration begins manifestly has for its physical cause the fact that the air, in entering the cavern, passes through an opening which in relation to the size of the cavity may be regarded as narrow, and which is often further diminished in diameter by mucous secretions; it is lost at the instant in which powerful inspiration effects the dilatation of the orifice, and in its place we have either bronchial respiration or râles caused by disturbance of the fluid lying in the cavity.

The metamorphosing respiratory murmur is usually heard only when respiration is vigorously carried on, seldom when it is tranquil and easy; its peculiar characters, also, are not always so pronounced as above described. It cannot even be considered a constant symptom of pulmonary cavity; it comes and goes, and varies to some extent, like the bronchial respiratory sound, ordinary râles, &c. But when it has once been clearly made out in any individual case, repeated and prolonged examination rarely fails to reveal its presence on subsequent occasions.

The fully-developed form of this murmur, as defined by Seitz, is rare; much more frequently there is, in excavation of the upper lobes, a respiratory murmur which resembles it thus far,—that it begins with a short, harsh, hissing sound, followed by râles and indeterminate respiration. If the simple fact that the inspiratory act consists of two sounds be held as sufficient warrant for designating it as a metamorphosing respiratory murmur, the relative frequency of the occurrence of this phenomenon is greatly increased.

To the category of bronchial respiratory sounds belongs

THE AMPHORIC RESPIRATORY MURMUR.

(The respiratory sound with a metallic tone or echo.)

By this name is understood a bronchial, rarely an indeterminate, respiratory murmur, accompanied by a tone of metallic quality or to which is added a metallic echo. It owes the appellation *amphoric* to the perfect analogy it presents to the sound produced by blowing into a pitcher or bottle.—This murmur occurs sometimes in inspiration alone, sometimes in expiration, at other times in both, but is almost always loudest and most distinct when accompanying expiration. Its fundamental character, as already mentioned, is generally that of the bronchial sound, the physical conditions which give to it its amphoric quality being precisely those which are most favourable to the development of bronchial respiration. It is only in very exceptional cases (see p. 141) that the *indeterminate* respiratory murmur is found attended by the amphoric echo.

The amphoric respiratory sound arises, as the production of an analogous sound by blowing into a pitcher teaches, only *in large pulmonary cavities* and in cases of accumulation of air in the pleural sac (*pneumothorax*). Its occurrence is subject to the following conditions:—

1. The *excavation* in the lung must be at least of the size of the closed fist, enclosed by walls of uniform density and thickness throughout, in free communication with one of the larger bronchi, and situated close to the surface of the lung. The first condition is a necessary one, as it is only in cavities of some considerable magnitude, possessing walls of uniform density, that the sonorous waves generated by the respiratory current of air can be reflected with that degree of regularity which is required for the formation of a really musical tone. Since, however, the amphoric quality is frequently wanting, even in cases in which the foregoing conditions are present, the explanation of the causation of the amphoric echo is incomplete without the addition of another factor, namely, *consonance*. The air-space of the cavern forms a resonance-chamber, in which the breath-sounds are intensified and, through the regular reflection of the waves of sound, so modified as to acquire a tone of distinctly musical quality.

The second condition,—free communication between the vomica and the bronchus leading to it, requires little argument in its support, as it is obvious that if the bronchus be blocked up the respiratory sound must disappear or become confused and indistinct; it returns, however, after repeated and forcible coughing, particularly when this is accompanied by expectoration.—The third condition,—the proximity of the cavity to the surface of the lung, is rarely absent when the vomicæ are of large size.

The clearness of the amphoric respiratory murmur is in no way influenced by the presence of a certain amount of fluid, as well as air, in the cavity. This may be shown by blowing, first into an empty pitcher, and then into one nearly full of water. It is essential only that the vomica do not contain so much fluid that the volume of the latter greatly exceeds that of the air-contents, as in such circumstances the respiratory murmur is usually completely hidden by abundant moist râles, and, more particularly, the existence of *consonance*, on which, to a great extent, depends the amphoric character of the breath-sounds, is rendered impossible.

As the great majority of pulmonary vomicæ occur in the *upper* lobes, so it is chiefly at the corresponding parts of the thorax that amphoric respiration is heard; it is loudest *anteriorly*, owing to the greater facilities offered by the front wall of the chest for the conduction of sound. In cases in which the cavities were very large I have observed it also on the *posterior surface* of the chest.

The metallic echo is always clear and acute, and its musical character often so marked that its pitch is accurately and easily determinable. Frequently several tones, the harmonic over-tones of the fundamental tone, are recognisable in it, and the impression then conveyed to the ear is precisely that given by the sweeping of the wind over the strings of an Æolian harp.—The amphoric echo may be the sole abnormal quality distinguishable in the respiratory sound, when there happens to be little or no fluid in the cavity, or when the patient breathes so feebly that the fluid is not agitated by the breath-current; but in the greater number of instances the amphoric respiratory murmur is accompanied by râles having a ringing metallic character (*metallic tinkling*, see p. 156).

At those parts where the respiratory murmur is amphoric the percussion-sound is also frequently modified in a similar way.

2. Amphoric respiration is also met with in *pneumothorax*, but

only when the lung is still able to expand so far as to permit the entrance of a certain quantity of air. This is possible only when the pulmonary fistula, through which the air reached the pleural sac, closes again relatively quickly; this rapid closure takes place in most cases, while persistence of the fistula, that is, the establishment of free communication between the air in the pleura and that in the bronchi and trachea, is the rarer event. If the bronchial fistula be obliterated, and if the lung be not completely collapsed, but still capable of a certain amount of expansion, a breath-sound may be heard at those points (usually posteriorly) which correspond to the position of the permeable portion of lung; this respiratory murmur is most commonly of the *indeterminate* variety, more seldom bronchial, and attended by a metallic clang or echo.

Here the explanation of the occurrence of amphoric respiration is that the sonorous waves borne along by the air entering the lung,—those constituting the ordinary respiratory murmur, are transmitted through the pulmonary substance to the air confined in the pleural cavity, throwing it into simultaneous vibration.

In certain rare cases, in which dyspnœa is the most prominent symptom, amphoric respiration is sometimes developed altogether independently of the existence of cavities. It arises in the pharynx, on making a deep inspiration with the mouth widely open, and may be conducted to the surface with such intensity as to be audible over the greater part of the upper thoracic region, more especially in the interscapular area. This variety of amphoric respiration is sometimes at once abolished by closing the mouth.—Friedreich asserts that in some very aged persons an amphoric respiratory murmur may be heard between the shoulder-blades, unconnected with any dyspnœa; I have never seen such a case.

INDETERMINATE RESPIRATORY MURMUR.

A large number of respiratory murmurs have not the characters of either vesicular or bronchial breathing; these, on Skoda's suggestion, are named *indeterminate* respiratory sounds. Strictly speaking, however, this category includes also all those transition-murmurs which, though somewhat undefined, are more or less distinctly bronchial or vesicular; these are designated, as formerly stated, as "somewhat vesicular," "obscurely vesicular," "obscurely bronchial," &c., the term indeterminate being reserved for those in which no trace of either fundamental quality is appreciable. The more practised the ear in the discrimination of the less-marked shades of difference between bronchial and vesicular respiration the more limited becomes the domain of the indeterminate respiratory murmur. It is those transition-murmurs which seem to present most difficulty to learners, the *truly indeterminate* sounds being as easily recognised as the purely bronchial or purely vesicular sounds.

As the indeterminate respiratory murmur resembles no other known sound it scarcely admits of description; a knowledge of its properties, therefore, is obtainable only by personal investigation and practice. A general conception of its qualities may be arrived at by auscultating the chest of a robust man, directing him at the same time to breathe exceedingly *superficially*, when the respiratory sound heard at those parts in which the muscles are well developed (the supra- and infra-spinous regions, for instance), will be found to be no longer vesicular but of the *indeterminate* class. The cause of this lies in the fact that in shallow inspiration the aerial current enters the alveoli with so little force or energy that the respiratory sound, in its transmission through the thick covering of muscles about the shoulder, is wholly deprived of its vesicular character; but on *deep* inspiration the indeterminate murmur becomes clearly vesicular. The gradual transition from vesicular to indeterminate breathing may be demonstrated by slowly increasing or decreasing the depth of each inspiration. An equally good method of exercising the ear in the study of the transition sounds is to auscultate the respiratory murmur at a point some distance removed from the part at which it originates,—as by passing the stethoscope slowly downwards from the borders of the lung into the hepatic region; the nearer the instrument is brought to the margin of the ribs the more obscure, the more indeterminate, becomes the respiratory sound.

As a pathological phenomenon the indeterminate respiratory murmur is of very frequent occurrence; it is sometimes persistent, at other times of short duration, confined to a limited area or diffused over a considerable extent of surface.—The causes giving rise to it may be arranged in the following groups:—

1. *Insufficient expansion of the alveoli.* This may be the result of diminution of their elasticity, as is so often observed in vesicular emphysema; or of infiltration of the air-cells (with fluid or plastic exudation), or of compression or atrophy of the

lung. In all these cases the volume of air which gains access to the alveoli is so small that the vesicular murmur is not developed,—in shallow respiration at all events. The intensity of the indeterminate sound is still further lessened when it traverses a mass of fluid, such as abundant pleuritic exudation. Effusions into the pleura are the most common conditions which are found to obstruct the free transmission of the breath-sounds; compression of the lung also, by air or tumours in the pleural sac, acts in the same way.

2. *Obstruction of one of the larger, or of several of the smaller, bronchi of an infiltrated portion of lung, by the presence of superabundant mucous secretion.* In such cases, as only a very small quantity of air reaches the vesicles, the respiratory murmur is indeterminate. This occurs extremely frequently as the result of the bronchial catarrh which accompanies infiltration of the lung-tissue. If the mucus be expelled by coughing, or shifted from one part of the air-passages to another, the bronchi of the part affected again become pervious, the indeterminate quality disappears from the respiration, and breathing becomes vesicular or bronchial according as the lung substance is still capable of expansion or is completely consolidated.

3. *All respiratory murmurs become indeterminate when masked by loud accessory sounds (râles)*; hence the frequency of indeterminate respiration in all cases in which the bronchi, alveoli, or pulmonary excavations are occupied by fluid secretion, the agitation of which by the breath-current gives rise to numerous râles. According to the distribution of the latter the indeterminate murmur is strictly circumscribed or widely diffused. Free expectoration also, or vigorous coughing, may so far alter or diminish the intensity of the râles that the true respiratory sound becomes again audible, this being vesicular if the lung still possess its normal spongy structure, bronchial if it be totally devoid of air. But in other cases, notwithstanding the diminution in the intensity and number of the râles, the indeterminate character of the breath-sound remains. In many instances a deep inspiration on the part of the patient causes the indeterminate respiratory murmur, on whatever condition it depends in the particular case, to become vesicular or bronchial,—vesicular, for example, in pulmonary emphysema, or bronchial in infiltration of the parenchyma; it is only in cases of very copious pleuritic

exudation that deep inspiration does not cause it to take on the vesicular character.

It is often possible to determine, and that with a considerable degree of certainty, to which of the above-named causes the indeterminate respiratory murmur owes its origin. They may all be in operation together: thus, in an infiltrated lung, the bronchi of which are also in a state of intense catarrhal inflammation, it is due to the slight amount of expansion of which the consolidated part is capable, to diminution in the quantity of air which reaches it (from tumefaction of the bronchial mucous membrane), and to the masking of the respiratory sounds by moist, catarrhal râles. In other instances, as in severe pulmonary emphysema without catarrh of the bronchi, or in cases of complete atelectasis of the lung-substance from the presence of a large pleuritic effusion, but *one* cause can be alleged for the indeterminate character of the breathing,—the insufficient expansion of the alveoli.—From these examples it may, with justice, be inferred that the indeterminate respiratory murmur possesses in general no precise diagnostic signification. Whilst the vesicular murmur or bronchial breathing at once discloses the condition of the subjacent lung, the former showing that it is still permeable, the latter that it is consolidated, indeterminate respiration *alone* gives no information of this kind, as even where it is heard the lung may or may not contain air; it is only on taking into account the other auscultatory phenomena that it may, in certain cases, be traced back with confidence to its physical cause. But occasionally, even when occurring *alone*, it may, if strictly localised, be sufficient to warrant a diagnostic conclusion. Thus, if it be audible and persistent in *one* apex, whilst in the other respiration has its ordinary vesicular character, it points decidedly to incipient condensation, to diminished expansibility from commencing disease in the part. Nevertheless, such instances (the occurrence of indeterminate respiration without accessory sounds) are rare, as the morbid process beginning in the apex usually excites also a catarrh of the finest air-tubules, giving rise to small, fine râles. Sometimes, however, an unaccompanied indefinite respiratory murmur may be observed in one apex when the disease has been arrested and to a certain extent cured, some condensation only remaining; here the catarrh has disappeared, and the râles, accordingly, are wanting.

RÂLES.

When the organs of respiration are perfectly normal the unmixed vesicular inspiratory and indeterminate expiratory murmurs are the only breath-sounds heard over the whole thorax; râles are entirely absent, as the mucous lining of the air-passages is everywhere smooth and secretes no more fluid than is just necessary to keep it moist. But as soon as this membrane becomes rough and uneven through swelling, and an augmented secretion of fluid takes place at any part of the bronchial ramifications, certain accessory sounds, named *râles*, are added to the respiratory murmur.

The manner in which these râles are produced in the larger and smaller bronchi, in their most minute subdivisions, and in the air-cells, varies very considerably. In bronchi of large calibre and in pulmonary vomicæ the passage of the respiratory current of air (both in inspiration and in expiration) through the fluid products, causes the formation of numerous bubbles which burst with an explosive, crackling sound,—like those rising in soapy water or other frothing or fermenting fluids, or in liquids which are forcibly shaken.* The disturbance of the fluid secretions also, by the breath-current, (which can obviously take place only within very narrow limits), may give rise to râles *without* the throwing up of bubbles, as in a body of fluid roughly shaken; and finally similar crackling sounds sometimes originate in bronchi in which no fluid is present, from vibration of the folds of the swollen mucous membrane. But in the bronchioles and in the air-cells the force of respiration is so slight as to be insufficient for the raising of bubbles in the secretions, and the alveoli, further, scarcely afford room enough; doubtless the râles here owe their existence to the circumstance that at the instant in which the smallest air-tubes and the air-vesicles are dilated in inspiration their walls are suddenly separated from their fluid contents to permit the passage of the current of air.

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* Talma has recently asserted, as the result of a number of physical researches conducted by him, that the bursting of bubbles is of itself noiseless, and that râles depend on the agitation of the fluid and the formation of a multitude of little projections on its surface; these little tongues vibrate and awake secondary vibrations in the mass of air pent up in the tubes which contain the fluid (in the case of the lung—the bronchi and cavities).

In expiration, on the other hand, when the vesicles shrink again to their former dimensions, the above-named cause is not in operation, and in fact râles are somewhat rare in this phase of respiration; but in those cases in which they are heard their occurrence may be explained on the supposition that the viscid, tenacious exudations offer considerable resistance at parts to the expiratory air-current, which, nevertheless, forces its way through them, and so breaks them up and agitates them again.

That the râles developed in the bronchioles and air-cells containing fluid secretions do not depend on the formation and bursting of bubbles, but on the rapid and violent separation of the agglutinated walls of these finer air-passages from each other, may be inferred from certain physical analogies and some physiological and pathological observations still to be mentioned; thus, a very similar sound is obtained by pulling asunder two fingers previously gummed together by their palmar surfaces, by applying the tongue to, and suddenly detaching it from, the roof of the mouth, or by inflating the collapsed lungs in the dead body (Wintrich), the walls of the alveoli being in contact after death. From the last of these examples, the sound generated in which bears a very close resemblance to a râle often heard in the living subject (the crepitant râle, to be subsequently described), an important diagnostic inference may be drawn: *that the râles originating in the air-cells and in the finest air-tubes do not necessarily depend on the presence of fluid secretions* in these parts, but that they *may sometimes arise from the sudden separation of the cohering walls of the alveoli, quite independently of the existence of any trace of exudation.*

Râles are classified as *moist* and *dry* according to their character, that is, according to the impression they convey to the ear. The difference results from the varying consistence of the bronchial secretions, the former variety being produced when it is *thin* and watery, the latter when it is *thick* and tenacious.

Moist râles most usually resemble the sounds caused by the breaking of bubbles in a fluid, such as the bursting of soap-bubbles in water, or of the bubbles which come to the surface of water beginning to boil; other, finer râles are more comparable to the sound elicited by rubbing the hair between the fingers, or to the crepitation of salt thrown on the hot-plate of a stove. But râles are subject to so much modification, and vary so considerably in character even in one and the same case, that to describe them fully by the common sounds of ordinary life is scarcely practicable, these latter being too few in number and not permitting of an accurate enough definition of the acoustic phenomena observed.

It is very seldom possible to determine, from the nature of the râles, the constitution of the fluid in which they are generated, that is, to affirm with precision whether it is serous, mucous, purulent, or sanguineous; such inferences are deducible only from a general knowledge of the character of the local morbid process.—But the seat of the râles, on the other hand, whether in the largest, medium-sized, or smallest bronchi, or in pulmonary cavities, may usually be ascertained with the greatest exactness, by a consideration of the various qualities of the râle, now to be described in detail.

1. Stage of Respiration in which the Râles occur.

Râles are audible sometimes in inspiration alone, more rarely in expiration alone, or during both inspiration and expiration. They usually appear at the acme of inspiration and at the beginning of expiration, when the fluid in which they originate is lodged in the finer bronchi. But if the secretion be so abundant as to occupy the larger as well as the smaller air-tubes, and if respiration be carried on with sufficient energy, the râles are heard not merely during the whole of the inspiratory period but also—though necessarily considerably enfeebled by the resistance offered to the escape of the expired air—throughout expiration; the rattling sound is then, in fact, almost *continuous*. The best example of this condition is found in diffuse bronchitis, though even here the râle is only temporarily, and not at every examination, continuous. Violent coughing, accompanied by expectoration of mucus, deprives it of its continuous character for a longer or shorter period.

Sometimes râles occur even in the *respiratory pause*. These are described as *postexpiratory râles* by Baas, their peculiarity being that they follow, or prolong, the expiratory râles, whilst the chest-walls are in a state of absolute repose.—I have not unfrequently observed this postexpiratory râle, in cases of large pulmonary vomicæ containing much fluid, and am of opinion that its production may be most consistently explained on the assumption that the commotion set up in the contents of the cavity by the respiratory air-current does not at once subside, but goes on for a short time in certain small portions of the fluid. It can be shown by a simple experiment,—watching the bubbles that form on the surface of soapy water shaken up in a vessel,—that the bursting of bubbles in a fluid does not begin and end precisely with the act of agitation, but continues after the vessel is at rest.—I have not noticed the postexpiratory râle in any other conditions than those mentioned,—large pulmonary cavities.

2. AMOUNT OF THE RÂLES.

Râles may be *abundant* or *scanty*. Their abundance depends on the quantity of fluid in the bronchi, air-cells, or cavities, on the proximity of the affected part of the lung to the surface, (by which the explosion of every individual bubble is transmitted more or less accurately to the ear), and on the violence of the agitation of the secretions by the aerial current. Numerous râles, therefore, always indicate free communication between the diseased part of the lung and the bronchi leading to it; and if this be interrupted temporarily by the impaction of mucus in the air-passages, they are either abolished or become very scanty, even though the parts be loaded with fluid secretions.—Very abundant and persistent râles are designated as *gurgling*, from their resemblance to the bursting of large bubbles in a viscid substance. These present themselves most frequently in pulmonary vomicæ of considerable size containing a large quantity of fluid, and occasionally also in the smaller bronchi when these are filled with catarrhal secretions.

The less the amount of fluid the scantier become the râles and the stronger the inspiratory effort necessary to produce them. Occasionally they are so scanty that only a few bubbles form or burst during each inspiration, whilst during several consecutive respiratory acts absolutely none may be heard, or they may become appreciable only after prolonged coughing. A very scanty râle often disappears completely when the patient breathes deeply and strongly for some time, as when undergoing examination by auscultation. An extremely scanty râle, consisting, for instance, of but one or two isolated bubbles, appearing only after deep and forcible inspiration, is not necessarily dependent on the presence of fluid in the air-passages or alveoli, but may be caused by the rapid and violent separation of the adhering folds of the mucous membrane lining the bronchioles, or by the distension of collapsed alveoli. Perfectly healthy persons may thus, on making a sudden and deep inspiration, present a few scanty râles at various parts of the lungs,—in the apices, the lower borders, and some other regions; these, however, are lost at the next inspiration. In other respects they do not differ from the scanty râles which characterise the earliest stages of catarrh in the apices; a knowledge of the fact, therefore, that these sounds are sometimes produced in the above-mentioned way becomes of the greatest importance, as one is always tempted to refer the slightest trace of a râle in the summits of the lungs to commencing catarrh.

3. INTENSITY (CLEARNESS, LOUDNESS) OF THE RÂLES.

Râles show very marked differences in *intensity* in different cases, and are very variable even in one and the same individual. Their intensity is the greater the more abundant the secretions, the more energetic the act of respiration, the wider the lumen of the bronchi within which the fluid is accumulated, and the closer the affected part of the lung to the thoracic parietes.—The more abundant the fluid the more numerous the bubbles formed by each draught of air and the greater the total volume of sound produced. Râles are similarly intensified also by energetic respiration, as by this means more bubbles, and those frequently of larger size, are thrown up on the surface of the fluid. It is for this same reason, further, that râles occurring in the principal bronchi are louder than those formed in the smaller air-passages. There are certain râles, however,—such as those arising in the larynx, trachea, or bronchial trunks,—which, though they may be, and commonly are, so intense as to be audible even at some distance from the patient, (a phenomenon known as "a rattling on the chest" among the laity), yet do not necessarily indicate the presence of any very large quantity of fluid in those parts; it is but seldom, also, that râles generated in the finer ramifications of the bronchial tree, even though exceedingly numerous, are observed otherwise than by mediate or immediate auscultation, and in these exceptional cases they are feeble. Râles originating in pulmonary excavations, though extremely loud as heard through the stethoscope, are never so intense to the ear held away from the chest as those produced in the large bronchi.

Râles, finally, are invariably stronger the nearer their place of origination to the part to which the stethoscope is applied. They are all capable of transmission through the tissues to points more or less remote, the loud being carried farther than the feeble;

we are never, therefore, warranted in concluding that the spot at which the râle may be detected corresponds precisely to the part at which it takes its origin. Very loud râles,—those met with in the right lung, for example,—may be propagated, though feebly, as far downwards as the hepatic region. But the ear, with a little practice, soon learns to distinguish with sufficient readiness between râles arising immediately under the auscultated spot and those which are transmitted thither, whether from adjoining or more deeply-seated parts of the lung. Transmitted râles are always *scanty*, as only the loudest of the bubbling noises, not *all* of them, are conducted to any distance; they are also *feebler* and *duller*, in this respect showing themselves subject to the same laws as other sounds. If râles of nearly equal intensity be heard over a large extent of surface, it may be inferred that the cause producing them is in operation at all parts throughout the whole of the space indicated; if at different, opposed parts of the thorax,—on the right and left sides, for instance, they belong to both lungs, as the conduction of râles from one lung to another is rare, taking place only when they are exceedingly loud and abundant in the region in which they are set up.

Galvagni has stated that râles developed in the *deeper* parts of the lungs are best heard *through the mouth*, in the cavity of which they are considerably intensified by consonance, and that they lose in intensity in transmission through the spongy lung substance (which is of very low conducting power) and the thoracic parietes.

I have been led, as the result of a long series of original investigations, to modify this view considerably. It is perfectly true that certain râles, those in the apex, for instance, appear surprisingly loud and clear if the trumpet-shaped extremity of the stethoscope be placed in, or even close to, the open mouth of the patient; in this way dull, indistinct râles acquire a clear resonant character, while those which are already of this nature become exquisitely metallic. Râles, therefore, are intensified by consonance in the *buccal cavity*, as the same sound is heard much more feebly over the larynx than at the apex of the lung. But I have never been able to convince myself that râles arising in the deeper and lower portions of the lungs, sounding faint and scanty on the surface of the chest, are louder and more numerous when auscultated in the mouth, though I have observed that dull, non-resonant râles originating even in the lower lobes, may be audible in the mouth, and assume there a ringing character.—These methods of auscultation obviously do not enable us to decide whether a râle comes from the right or left lung, from the upper or lower lobe, &c.

4. Size of the Râles.

Different râles convey very different impressions to the ear with respect to *the size of the bursting bubbles.*

The size of the bubbles is determined by the quantity of fluid present and the strength of the current of air passing through it, above all by the lumen of the bronchi in which the râles are developed.

Râles are divided, according to the apparent size of the bubbles, into *fine bubbling* and *coarse bubbling râles*, those of intermediate magnitude, or which present a combination of the two preceding varieties, being designated as *medium-sized bubbling râles.*

The *fine bubbling râles* are generated for the most part in the smaller and smallest subdivisions of the bronchi, the *coarse bubbling râles* in the larger bronchi; but even in the largest and widest parts of the air-passages very fine moist râles may arise. If the fine and coarse bubbling be combined,—a condition readily detected by the ear,—they are termed *unequal or irregular bubbling râles;* if, on the other hand, the bubbles seem to the ear to be equal in size, they are named *uniform bubbling râles.*

Among the *fine bubbling râles* there is one variety which demands special attention. It originates in the extremities of the bronchioles and in the air-cells,—air-spaces of the very smallest capacity; the bubbling of which it consists is therefore exceedingly minute; as the alveoli, also, are all equal in diameter the individual explosions are of uniform size. Such a râle is spoken of as a *fine, uniform bubbling râle;* it is known also as the *moist crepitating* râle of Lænnec, from its resemblance to the sound produced by rubbing the hair of the head, especially in front of the ear, between the fingers. It is usually only at the acme of inspiration, seldom at the beginning of expiration, that it is heard.

When crepitation is distinctly appreciable we have proof that the air-vesicles still admit air, and the proper diagnostic inference with regard to the state of the pulmonary parenchyma is simply that the alveoli contain both air and fluid. This condition occurs in the most pronounced form in the first and third stages of pneumonia, so that uniform crepitation may be considered as almost pathognomonic of the disease in these stages.

Crepitation in which the bubbles are uniform must be sharply distinguished from that in which they are *irregular* in size. The latter is specially characterised by the fact that in it not only fine and regular bubbles, but also some others of much larger size, are heard. Diagnostically this distinction is of some importance, as the genuine uniform crepitation of Lænnec is developed in lung-substance which is still permeable to air, the irregular form very frequently in consolidated tissue, that is, not in the air-cells, but in the finest bronchi or their terminations.

Pneumonic crepitation lasts, provided there is no obstruction of any of the larger bronchi leading to the infiltrated part,—a condition which would weaken or abolish the râle,—till the alveoli are so completely filled with fluid that the air is entirely excluded; it returns as soon as the fibrinous exudation begins to be absorbed and the air once more gains admission to the vesicles.—Crepitation is also *usually unaffected by coughing*, as the contents of the alveoli cannot in this way be dislodged.

In pulmonary œdema, also, a serous exudation,—not fibrinous, as in pneumonia,—is poured into the air-cells, so that here too are furnished the conditions necessary to the existence of the crepitant râle,—the intimate intermingling of air and fluid; but in this instance the râle is not so well defined as in the forementioned stages of pneumonia, as the *bronchi* also are filled with fluid, and the *irregular bubbling râles* produced in them being added to the crepitation proceeding from the alveoli, the general impression given to the ear is no longer that of the *uniform* crepitant râle as in pneumonia.—In the catarrhal affection of the most minute bronchi which usually attends the various diseases of the lung-substance, a few *non-uniform*, but nevertheless very fine, crepitations may be heard here and there over the chest, and these may be of merely temporary duration or persistent.

Crepitation is also occasionally audible for a short time in collapsed conditions of the lung, in those, for instance, in which the organ has been for a long period subjected to pressure by pleuritic effusion, or in the retracted pulmonary parenchyma situated above the level of a pleuritic exudation; here it may possibly be the result of a catarrh of the extremities of the finest air-tubes,—a condition often associated with pleuritic exudation, —though it frequently arises, altogether apart from the presence of fluid in the bronchioles, solely from the tearing asunder of the collapsed and adherent alveolar walls (compare p. 146).

There is a very deceptive kind of *artificial crepitation* which must be guarded against in auscultating the thorax. In those in whom the skin on the front of the chest is plentifully covered by hair each inspiration is accompanied by a sound the exact counterpart of the crepitant râle, produced by the rubbing of the little crisp hairs on the end of the stethoscope. This noise is often so loud as to interfere seriously with the perception of the true respiratory sounds. It is a source of error, however, which may be completely eliminated by simply wetting the hairs with water, when they apply themselves flatly and closely to the surface of the chest.

The *medium-sized bubbling râles*, by which is meant, as already noticed, the transition-form between fine and coarse râles or the sound which results from the mixing of these, is set up in bronchi which differ considerably with respect to calibre, but not in the very finest or in the very largest; in the smaller air-passages the fine bubbles greatly outnumber the large ones, in the larger bronchi the reverse is the case. In pulmonary excavations, also, in which there is a very abundant fluid secretion, the râles are usually composed of bubbles of medium size.—These râles may be still further subdivided into two classes, according as the bubbles are *over* or *under* the *average* size,—a degree of refinement which, as it depends almost entirely on subjective appreciation, is of no practical value.

Coarse bubbling râles occur chiefly in the principal bronchi and in the trachea, and are usually loudest and most prolonged in expiration; to this category belongs the death-rattle in the air-passages of the dying. The ability to distinguish between medium-sized and coarse bubbling râles,—a point of no great diagnostic importance, is easily acquired by the ear, with a little practice, while the difference between fine and coarse râles is so obvious as to strike every one at once.

The medium-sized and coarse râles, which are always louder than the fine râles, are found not only in simple primary catarrh of the bronchi, but also in the secondary bronchial catarrh which accompanies the parenchymal diseases of the lungs, and in pulmonary cavities. These râles, therefore, can have but one diagnostic signification,—that fluid is present in some of the larger air-passages; but without a consideration of the character next to be discussed, (resonance or non-resonance of the râles), and of the properties of the respiratory sound, such phenomena alone are not sufficient to indicate whether the tissue of the lung is still freely permeable to air or is consolidated.

Skoda has for this reason applied to the whole group of râles just described the collective term *indeterminate* râles, regarding them as analogous to the indeterminate respiratory murmur, as neither class of phenomena offers any indication of the condition of the lung-tissue with respect to air-contents. Practised auscultators, nevertheless, are able to determine, from certain peculiar qualities of the non-uniform bubbling râles, whether they are due to a simple catarrh, or to the secondary catarrh dependent on condensation of the lung-substance. No general rules can be laid down for guidance to the formation of such a diagnosis, which is, indeed, rather a matter demanding skill and experience; it must suffice, therefore, to point out some of the principal differences between the râles of primary and of secondary catarrh. In simple bronchial catarrh the râles are heard over the greater part of one or both lungs, they vary greatly in intensity at different points, consisting of bubbles of very unequal size, and are usually accompanied by dry râles (see p. 158); the vesicular inspiratory sound, further, is generally of the rough, harsh type, and a characteristic thrill is often felt by the hand laid on the chest-wall. These details are applicable also to the bronchial catarrh associated with pulmonary emphysema. But the râles of secondary catarrh, catarrh consequent on condensation of the pulmonary parenchyma, are more limited in distribution, being frequently confined to small circumscribed spots; they are usually much finer,—as such catarrh most commonly has its seat in the smaller bronchi,—are more seldom accompanied by dry râles, and are very rarely, and then but feebly, appreciable to the hand placed on the surface of the chest.

5. RESONANT AND NON-RESONANT (TONELESS) RÂLES.

Resonant (or sonorous) râles are those in which a tone of somewhat musical quality is distinguishable, the non-resonant those which make on the ear the impression only of a non-musical sound or noise. The former usually appear to the ear *clear* and *high*, the latter *dull* and *low in pitch*; in well-marked, typical instances, therefore, the distinction between the two varieties is easily made, while in the transition-forms, which are designated, on the one hand, as *obscurely* or *approximately resonant*, or on the other as *almost toneless*, much has to be left to the subjective appreciation of the individual examiner.—All fine, coarse, or medium-sized bubbling râles, with the exception of the small *uniform* râle (the crepitant râle), may become resonant under certain circumstances described below.

Resonant râles arise only in consolidated lung-tissue and in cavities, and should therefore be interpreted in the same way as bronchial respiration; on the other hand, râles which originate in parts of *spongy* texture are invariably *non-resonant*.—The cause of this resonance is to be found, not in Skoda's assumed consonance,* but in the favourable conditions offered by the dense tissue for the transmission of each individual sound to the surface. As the râle generally loses its resonant quality in passing through expansible, air-containing lung, it is usually toneless at some distance from its point of origination.—If in listening to a resonant râle special attention be directed to *individual* bubbles, it will be found that they do not all burst with the same musical sound, that many, in fact, are absolutely non-resonant; the more numerous the bubbles having the sonorous character the more distinct is the resonance; this resonance is also greater the more complete the consolidation of the lung, the closer the indurated part lies to the surface, and the larger the bubbles thrown up in the fluid. But it is far from being invariably the case that the râles heard in condensed lung-substance are resonant; they are sometimes perfectly devoid of the sonorous quality, this being specially apt to occur if the dense part is small, or if between the portions of solidified tissue the lung is still quite spongy in texture.

But frequently, with well-marked physical signs of consolidation,—percussion-dulness and loud bronchial respiration,—the râles may appear non-resonant, whilst at a subsequent examination they may again be distinctly resonant; this change does not in all cases admit of ready explanation.—The most clearly resonant râles are those generated in cavities situated close to the surface of the lung and surrounded by dense, firm parenchyma.

Thus, whilst *resonant* râles indicate with certainty the presence of *condensation* or *excavation* of the lung-tissue, *non-resonant* râles *do not necessarily exclude* the existence of these conditions. In the absence of the resonant râle the diagnosis of consolidation of the lung-substance rests on the results of percussion (dulness), and of auscultation of the respiratory sound (bronchial breathing). In those parts in which the râles are of resonant quality the respiratory murmur may be bronchial, or amphoric, or indeterminate, but obviously *never vesicular.*

The non-resonant râles, yielding, as they do, no direct evidence as to the condition of the lung-substance, fall naturally into the category of indeterminate râles already mentioned.—Sometimes, however, they bear no resemblance whatever to the usual sound of bursting bubbles, being of a peculiar *creaking* or *crackling* quality. Such a râle, which constitutes the transition-form between moist and dry râles, indicates great swelling of the bronchial mucous membrane, and a scanty or very tenacious secretion. These creaking râles occur very frequently both in primary bronchial catarrh and in the catarrh which accompanies chronic induration of the lung.

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* Skoda names the resonant râle the *consonating* râle, in conformity with his view of its origin.

As the apices of the lungs are the parts most commonly affected by condensation it is here that the creaking and crackling sounds,—more frequently the former,—are oftenest heard. These peculiar phenomena are observed only when the râles are scanty; they appear, also, to originate in the immediate vicinity of the ear, in the most superficial parts of the lung.—After remaining audible for a considerable period they may completely disappear, on account of diminution in the quantity of the bronchial secretion. In general their characters are subject to much variation, like those of all the other moist râles.

METALLIC RÂLES.

The most exquisite form of the resonant râle is termed the *ringing, metallic râle.*

These râles are accompanied by a tone of genuine musical quality, very clear and of accurately determinable pitch. Frequently all the bubbles formed by the agitation of the fluid secretions have this ringing metallic character; in other cases only a few of them possess it, while the remainder are obscurely resonant; and in still another class of cases the râle, especially when it consists of but a few bubbles altogether, gives the impression as of drops of fluid falling from a height with a sound of acutely metallic intonation.

Râles of metallic timbre, especially single bubbles, may be best imitated by causing drops of water to fall some distance to the bottom of a metallic vessel.

Lænnec employed the term *metallic tinkling* to designate the phenomenon just described,—the intensely metallic sound of single bursting bubbles, simulating the dropping of water, occurring in large pulmonary excavations or in pneumothoracic cavities occupied by a certain quantity of fluid. It is certain, however, that the sound is exceedingly deceptive; it is not produced by the dropping of fluid, but, as above stated, by the explosion of single bubbles, whose tone is intensified by resonance in the cavity.

Râles of metallic timbre arise exclusively in cavities of considerable size, as large, at least, as the closed fist, which are enclosed by walls of uniform density, and are situated near the surface of the lung. These are precisely the conditions enumerated as necessary to the occurrence of the metallic percussion-sound and of the amphoric respiratory murmur. But the free communication which, in order to the development of amphoric respiration, must exist between the air of the cavity and that of the bronchus leading to it, and between the latter and that of the trachea, is not an indispensable condition in the case of the metallic râle; this is obvious from the fact that in the somewhat rare cases in which there is absolute closure of the bronchus opening into a large cavity,—as indicated by the total suppression of the respiratory murmur,—the ringing metallic râle may be at once elicited by sharp coughing, when the fluid and air in the excavation are thrown into violent agitation. The *metallic* râle has been justly explained as a true *phenomenon of consonance*, in contradistinction to the simply resonant râle, which owes its peculiar qualities to the better conduction of sound through solidified tissue. Amongst the many resonant bubbles which go to constitute it, but few have the pure *metallic* resonance; these are they whose pitch corresponds to the proper tone of the cavity, and for them the cavity acts as a resonance-chamber, in which the ringing quality of the râle is intensified (compare Bronchophony, p. 169). By using a suitable resonator, also, (see p. 95) the metallic sound may be greatly increased in intensity. It is often possible to recognise in the metallic râle several tones (fundamental tone and overtones) standing in harmonic relation to each other.

In those cases in which this râle is heard the respiratory murmur,—when not temporarily suppressed or rendered indeterminate by plugging of the principal bronchus, in which circumstances the metallic râle also is wanting during the respiratory act and is audible only on coughing,—is *amphoric* (accompanied by a metallic echo), so that amphoric respiration and metallic tinkling have come to be shortly designated metallic phenomena, or, as they occur almost exclusively in cavities, as *cavernous phenomena*.

THE SOUND OF SUCCUSSION IN PYO-PNEUMOTHORAX.

There remains for discussion a special variety of the metallic râle,—a *splashing*, accompanied by a ringing sound, heard in cases in which the *pleural sac is occupied by air and fluid* (pyo-pneumothorax), on shaking the patient while the ear is applied to his chest. This method of exploration was practised by Hippocrates,—hence the name by which it is generally known, *Hippocratic Succussion*. On shaking a jug partially filled with water a fair representation of this sign is obtained. In some cases it is very feeble, appreciable only by immediate auscultation or through the stethoscope, whilst in others it is audible even at a considerable distance.

In one patient who was under my care, suffering from pyo-pneumothorax on the left side, the splash could be heard in all parts of a large class-room, when he raised himself quickly on his toes and dropped back again on his heels.

When the cure of a pneumothorax is effected by the outpouring of a pleuritic exudation which, by its increase, gradually takes the place of the air, the sound of succussion is no longer appreciable. In encysted pyo-pneumothorax also, especially if it be of limited extent, the splash is wanting; and, further, it does not appear to be present even in every case in which the effusion is free and unconfined by adhesions, but only in those in which it is also of very fluid consistence (serous), and in that way easily thrown into commotion by shaking the patient.

The splashing succussion-sound *may* also be observed in *pulmonary vomicæ*, when they are of the *size of the closed fist* at least, and contain a very abundant and *thin* fluid secretion; but as the latter condition is exceedingly rare in phthisical and bronchiectatic cavities, it is only in very exceptional cases of this kind that the sound in question is heard. Gangrenous cavities do certainly contain a secretion of this character, but are seldom so large as to form a resonant chamber sufficiently spacious for the proper development of the splashing sound.

DRY RÂLES.

By this term are meant râles which do not create the impression of the bursting of bubbles or of liquid in motion, but which are comparable rather to the whirr of a revolving wheel, to the crisp noise produced by walking on frozen snow, to the crackling of a dried bladder, &c. These comparisons come far short of exhausting the varied characters of the sounds under consideration; they cannot be regarded as minutely descriptive of them, but merely as expressive of some of their most striking and prominent resemblances. A little practice, however, will soon enable any one to distinguish readily between moist and dry râles. The transition from moist to dry sounds is gradual; the *creaking* and *crackling* sounds described on p. 156 may be taken as representing the forms intermediate between the two definite types, though they are almost entitled to be looked upon as dry râles.

The general diagnostic inference to be drawn from the presence of dry râles, with reference to the condition of the bronchi, is that *the mucous membrane is swollen,* and covered by a *scanty, exceedingly viscous secretion.*

From these dry râles is to be carefully distinguished still another class of sounds, in which there is no longer to be found any trace of the characteristic features of the râle; these are known as the *snoring*, *hissing*, and *whistling* sounds. As the sensory impression produced by them corresponds exactly to that indicated by the names they bear, (the sonorous rhonchus, for instance, resembling closely the deep tone of a bass string), their recognition presents no difficulty. These sounds are caused by the passage of the respiratory current of air through bronchi whose calibre is encroached-upon by swelling of the mucous membrane, or which are at parts temporarily obstructed by plugs of mucus. These conditions alone are enough to render the respiratory murmur loud and harsh, as may easily be shown experimentally by blowing through caoutchouc tubes which are compressed at certain points; but besides these causes the vibration of the prominent folds of the swollen mucous membrane and of the tough secretions adherent to them, take an important part in the production of the sounds. The *snoring sounds* (sonorous rhonchi) originate in the *large and medium-sized bronchi*, the *hissing* and *whistling* sounds (sibilant rhonchi) in the *small and smallest air-passages.* The former are low-pitched, the latter high; and this is exactly in accord with the teaching of physical science,—that the pitch of a tone, like that of a râle artificially produced in tubes, rises as the diameter of the chamber in which it is generated diminishes. These rhonchi are very common in chest affections, and are most marked in acute and chronic bronchial catarrh,—both in the primary form and in that which results from the various diseased conditions of the pulmonary parenchyma, especially emphysema; they are generally audible over a large part of one or both sides, sometimes even over the whole, of the thorax, and vary in intensity with the degree of the swelling of the mucous membrane and the lumen of the bronchi attacked by the catarrhal process. The loudest are the sonorous rhonchi, occurring in the large bronchi. The character of the sounds depends on the size of the bronchi involved, being snoring or hissing and whistling according as it is chiefly the large or the small air-tubes that are affected; occasionally all three varieties are heard simultaneously, at the same or at different parts of the thorax. They are considerably louder than the moist râles, and are appreciable, even at a distance from the patient, in both inspiration and expiration, sometimes throughout the *whole* of the respiratory act, as sighing, moaning, or cooing sounds. As in these circumstances the elasticity of the lungs is diminished and expiration prolonged, the duration of the expiratory rhonchi is much longer than that of the inspiratory sounds. If the bronchi contain also a certain amount of fluid secretion moist râles are added to the dry sounds, and as fluid is found more frequently in the larger than in the smaller bronchi it is the sonorous rhonchus which is most often accompanied by râles (of the coarse or medium-sized bubbling variety), whilst the sibilant rhonchi are commonly free from such indications of the presence of liquids in the air-passages. The respiratory murmur is sometimes audible along with the sonorous or sibilant sounds; it is vesicular so long as the lung-substance remains permeable to air, or may disappear entirely, as in aggravated cases of pulmonary emphysema; it may be completely masked by the loudness of the dry râles, or may possess the characters only of indeterminate respiration.—All these dry sounds, which are shortly designated as *catarrhal*, may be modified by the assumption of the ringing, resonant quality, when the lung-substance at the affected part is completely consolidated; it is scarcely justifiable, however, to base the diagnosis of consolidation on this sign *alone*, as even while the lung is perfectly expansible these rhonchi show a greater tendency than any of the other râles to take on the character referred to; this is clearly indicated in the names applied to them, sonorous, sibilant, &c. It is only necessary to auscultate a few cases of severe emphysema attended by dry catarrh, to become convinced of the frequent occurrence of this almost musical property of the dry catarrhal râles. They are all, especially the sonorous rhonchi, appreciable on the surface as palpable fremitus (see p. 68), but may also become unrecognisable, either by auscultation or palpation, for a longer or shorter period, or at least suffer considerable diminution in intensity, when the patient succeeds in expectorating some of the mucous secretions, which in these cases are always scanty and tenacious.

Several of the moist and dry râles, the special characters of which have been described, may co-exist in the same individual. Or the different orders of râles may assume the most diverse forms, merging into each other through multiform modifications, and that not only at different stages of the morbid process but within very short periods of time, in the space of a few minutes, for example, on coughing sharply. In the same way similar râles may be observed accompanying very different respiratory murmurs; thus with vesicular respiration all the râles, excepting the resonant and the metallic varieties, may be heard, with indeterminate respiration râles of every description, without exception, and with bronchial respiration râles of all kinds except the genuine uniform crepitation.

PLEURITIC FRICTION-SOUND.

This phenomenon presents to the ear much the same characters which are felt by the hand in Pleural Fremitus (p. 66). It gives the sensation sometimes as of the lightest rubbing, if stronger, that of shuffling, grating, or *creaking*, &c.; in the latter form, which is the most common, the sound is divided into several distinct portions of different intensity. It is sometimes audible only at the height of inspiration and the beginning of expiration, at other times, especially when breathing is vigorously carried on, throughout the whole of both phases of respiration. It is also generally, though not always, possible to form some preliminary conception of the intensity which the sound should present to auscultation from the distinctness of the thrilling sensation communicated to the hand. In some cases it is only its rougher, or more striking features that are thus perceptible to the sense of touch, whilst the ear takes in not merely these,

AUSCULTATION OF THE COUGH.

The cough of the patient is sometimes made use of as an aid to auscultation, but in many cases is also itself made the subject of direct examination.

As an adjuvant to auscultation it answers many purposes:

1. After repeated coughing inspiration becomes deeper and the respiratory murmur louder.

2. Casual obstruction of the bronchi by plugs of mucus is removed by coughing, especially if followed by expectoration; communication between the air-passages and the substance of the lung is thus re-established, and the respiratory murmur, previously suppressed or indeterminate in character, becomes clearer. The nature of the respiration after coughing depends on the state of the lung itself, being bronchial in consolidation or excavation of its substance, vesicular if it be still of spongy texture.

3. Râles are often first called into existence, or are considerably intensified, by coughing; it agitates the fluids which are lodged in the pulmonary vomicæ and in the bronchi, throwing them into more violent commotion than ordinary respiration; it frequently also has the effect of collecting and driving the secretions into more confined spaces, and thus of increasing the number and intensity of the râles. During the cough, therefore, and in the deep inspirations following it, the râles are heard with great distinctness. Occasionally, however, the râles, notwithstanding the complete absence of expectoration, become weaker at certain parts after coughing, and stronger at others; this is plainly due to the shifting of the liquids in the air-passages.—These changes in the râles, as the result of coughing, are observed with great frequency in diffuse bronchial catarrh.

4. The ringing character of the metallic râle is generally brought out by coughing, if it is not already observable in ordinary respiration.

The cough itself is auscultated, as it developes certain peculiar acoustic phenomena in consolidated lung-tissue and in cavities: it becomes exceedingly loud in solidified lung, louder, at least, than in lung which is permeable to air, and in large superficial cavities it further acquires a distinct *metallic ring*. Auscultation of the cough alone, therefore, is sufficient to decide whether the subjacent part of the lung is solid, of spongy texture, or hollowed out into cavities.

The cause of the greater distinctness of the cough over condensed tissue and excavations lies in the superior sound-conducting power of indurated lung-substance.

It remains now to discuss *the cough as a symptom* in diseases of the respiratory organs and *its signification from a diagnostic point of view*.

Cough is produced by the sudden and forcible opening of the glottis by the expiratory current of air. It is frequently an entirely voluntary action; at other times it occurs independently of the will, as a *reflex* action, when the terminations of the sensory nerves in any part of the mucous membrane lining the air-passages, from the larynx down to the smallest bronchioles, are subjected to any abnormal irritation. The most sensitive part of the mucous membrane is that which protects the larynx (especially the inter-arytenoid region) and the trachea as far downwards as its bifurcation; coughing may at any time be artificially excited in animals by irritating any part of this portion of the membrane (Nothnagel, Kohts). The mucous lining of the bronchi is much less sensitive. These views find ample corroboration in certain pathological observations; thus it commonly happens that most violent coughing is set up on the intrusion of foreign bodies into the larynx, and that, further, if the efforts made to expel these bodies be unsuccessful, if they rather sink deeper into the bronchi, the coughing becomes less distressing and may finally entirely cease.—Irritation of the costal pleura, also, provokes coughing, while stimulation of the visceral portion is powerless to produce this effect, as has been shown by experiments on animals and some observations in the human subject. Whether coughing may be brought on, by reflex action, from irritation in the stomach, is still doubtful, though some of the phenomena met with in disease seem to point in that direction; it has never yet been accomplished experimentally.—Stimulation of the vagus in the neck, of the superior laryngeal nerve, and of the nerve-centres from which the constituent fibres of the vagus spring, induces attacks of coughing (Kohts); in those cases, therefore, in which coughing appears as a symptom accompanying some disease of the nervous system, and apart from the existence of any concurrent pulmonary disorder,—as in some instances of hysteria,—it may be referred to an abnormal condition of irritation in the course of the vagus. Further evidence of such a purely neurotic origin is found in the fact that the cough usually comes on in paroxysms, like that of tussis convulsiva.—Most attacks of coughing are preceded by a *tickling* sensation in the larynx, even when the affection which excites them is not located at that point, but in the bronchi. This tickling forces on the cough, or at least makes it difficult to refrain from endeavouring to find relief in that fashion.

The irritation necessary to the provocation of coughing, apart from the accidental admission of foreign bodies into the larynx, is furnished in diseases of the respiratory organs by the swollen condition of the bronchial mucous membrane, or the presence of fluids in the air-passages,—such as bronchial mucus, inflammatory exudations, extravasated blood, pus, or serum; occasionally, also, simple engorgement of the lungs with blood, independently of any change in the mucous membrane, appears to be sufficient to cause coughing. Inasmuch, therefore, as almost all the diseases of respiration are, at some stage or other, attended by bronchial catarrh, localized or diffuse, it is plain that in almost all these affections coughing can scarcely fail to be a more or less prominent symptom, lasting either throughout the whole of the disorder, or developed only at some particular part of its course. Coughing is thus a symptom of the greatest importance in many diseases, especially in incipient phthisical affections, as, in the absence of other indications, it is not unfrequently the first sign which directs the attention of the medical attendant to the lungs.

Complete absence of cough in pulmonary diseases, either during certain longer or shorter periods or during the whole of their course, is exceedingly rare; the most probable explanation of such an occurrence is that in these cases the sensitiveness of the nerve-terminations in the bronchial mucous membrane is blunted. This may take place even under physiological conditions: in sleep, for instance, this irritability is much less marked than during the waking hours; and in certain pathological circumstances it may be similarly affected,—as when there is disturbance or enfeeblement of the sensorial activity, in collapse, and in profound exhaustion from disease.—But in the total absence of such morbid conditions, also, diseases of the respiratory organs may run their entire course without cough,—and it is these cases only which are above referred to as "exceedingly rare." Nevertheless even in these instances it cannot be said that cough is absolutely wanting, but only that it is so trifling, and that it appears at such long intervals, that it escapes the patient's notice.*

The *frequency* of the cough is variable: the intervals at which it occurs are very irregular, sometimes long, at other times short; at certain periods of the day, however, coughing is more frequent and violent,—in the evening, for example, especially in simple bronchial catarrh, or in the morning, shortly after waking from sleep, as in morbid processes of phthisical character. In general the frequency of the cough is directly proportionate to the intensity and distribution of the bronchial catarrh, increasing with each exacerbation of the affection and decreasing with the remission. Thus, emphysematous patients, and some others suffering from lung complaints, cough but little in summer, as the (secondary) bronchial catarrh almost disappears at that season of the year, while in winter both the disease and the cough return with all their former violence. Aggravation and mitigation of the cough, therefore, are signs which enable us to form at least an approximate opinion with regard to the progress of the original morbid process going on in the lungs, though they are obviously of such value only when the patient comes frequently under observation.

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* I have never yet met with a case of total absence of cough in lung disease. On careful cross-examination of the patient one rarely fails in obtaining from him an affirmative answer, even when the existence of cough is at first strenuously denied. Very many patients, also, do not regard a slight hawking from which they may suffer as a genuine cough.

The cough presents great diversity in respect to its special characters; these vary according to the *frequency* of the cough, its *intensity*, its *tone*, and whether it is *moist* or *dry*.

1. Each attack may consist of one or of only a few short, slight expiratory efforts;
2. Or of a series of several coughs, of somewhat greater intensity;
3. Or of a constant and rapid succession of violent paroxysmal seizures, broken only by a few deep, usually sonorous, inspirations.

The first variety, known in Germany by the name *hüsteln*, is met with most commonly in phthisical patients, and is regarded even among the laity as an ominous sign, though it is occasionally observed in diseases of a non-phthisical nature. This slight cough is generally not the only form which accompanies phthisis; it alternates very frequently with that described as the second variety. In particular, the cough which attacks the patient in the morning, soon after waking, is usually of a somewhat severe and persistent character, as the bronchial secretions which have accumulated in the air-passages overnight keep up a continuous irritation until expelled. Violent and prolonged coughing often excites vomiting, the sensory nerves of the stomach being irritated by the repeated and rough concussion.

The second variety of cough is the most common; it presents itself in all the diseases of the respiratory organs.

The third variety appears in its most typical form in tussis convulsiva, a disease of childhood which obtains its name from the nature of the cough. It occurs in distinct paroxysms of even several minutes' duration, separated only by a few deep, crowing inspirations; it is laborious and spasmodic, the countenance becomes darkly cyanotic, and the seizure not unfrequently ends in vomiting. In the intervals the fits of coughing are shorter and milder. In other diseases of the respiratory organs also the cough sometimes takes on this spasmodic character, but the attacks are then of shorter duration and not so typical.—The classification of the cough with regard to its *intensity* is embraced in the three foregoing categories; it is least marked in the first form (*hüsteln*), most marked in the spasmodic cough, and also, as in dry bronchial catarrh, when the secretions are very tenacious and therefore expectorated with some difficulty.

The cough is designated as *moist* or *dry* according as it is accompanied or not by expectoration. At the outset of any disease of the organs of respiration, especially bronchial catarrh, when the mucous membrane is still merely congested or swollen, but not yet covered by secretion, the cough is invariably dry; at a later stage it becomes moist. The expulsion of an offending portion of mucus is not generally effected by a single effort of coughing, and the process is the more difficult and troublesome the more deeply situated the part of the air-passages from which the secretion has to be brought.

The *tone* of the cough is exceedingly variable, and depends chiefly on the character of the cough in other respects,—whether it is forcible or feeble, moist or dry. The more violent the cough the clearer its tone, the feebler it is the duller its tone. It is also clearer when dry, and scantier when moist. The cough of laryngeal and tracheal catarrh, when very dry, is not unfrequently of a peculiar loud, clear character, (the "barking cough" of the laity). If the cough be dry and extremely laborious it is generally accompanied by a hissing sound, audible at some little distance from the patient, due to the forcible rush of air through the glottis; this same sound, moreover, is heard in every variety of cough, when auscultated by means of a stethoscope. It is a matter of daily experience also that the tone of a moist cough is subject to great modification by being mixed with the sounds caused by the agitation of the expectorated fluids.

From these considerations it will be seen that, apart from the very characteristic phenomena of hooping-cough, there is in general nothing in the nature of the cough that throws much light on the actual morbid changes going on in the lungs; at best it is only when taken in conjunction with the previous history of the case, especially as regards duration, that it assumes any diagnostic value. This diagnosis is most easily made in cases of chronic character, in which the principal question to be settled is simply whether phthisis exists or not; but even here the nature of the cough is not the sole, or even the chief, guide,—it should be supported by a minute physical examination of the chest, and an inquiry as to the presence or absence of the other phthisical signs, emaciation, &c.

It is not merely in diseases of the lungs themselves that cough is observed; it occurs also in affections of the pleura (pleurisy, for instance), but always in its less marked forms. In such cases it probably arises from irritation of the sensory nerves of the pleura, partly also from the slight bronchial catarrh which often attends inflammation of the pleura.—Cardiac diseases excite cough only when they set up secondary congestive catarrh of the lungs.

AUSCULTATION OF THE VOICE.

The vibration of the thoracic parietes, caused by the voice, and felt distinctly on the surface as pectoral fremitus, has already been discussed in the section on Palpation (see p. 63).

Auscultation of the voice is also frequently employed as a diagnostic aid in diseases of the organs of respiration, to supplement or confirm the results obtained by auscultation of the respiratory murmur.

When the respiratory apparatus is in its normal condition the words spoken by the person under examination are absolutely indistinguishable from each other, either through the ear applied directly to the chest-wall or through the stethoscope; the voice is heard merely as an undefined buzzing. The intensity of this sound at any part corresponds exactly to that of the vocal thrill; the conditions, therefore, on which depends the greater or less audibleness of the voice at any point on the surface of the thorax, are precisely those formerly enumerated as similarly affecting the intensity of the pectoral fremitus.

Pathological alteration of the voice, with respect to the distinctness with which it is conducted through the tissues to the surface, takes place in one of two directions: the voice may be rendered so feeble as to be totally inaudible; or it may be intensified to such a degree that a large number of the words become clearly articulate, and the auscultator has the impression as if the patient were speaking directly into his ear. Increase of the intensity of the voice is termed *bronchophony;* amphorophony (a metallic ring or echo produced by the voice), and ægophony (the bleating voice), are modifications of bronchophony.

ENFEEBLEMENT OF THE VOICE.

The sound of the voice, at the surface of the chest, is weakened or suppressed by the same conditions which lessen or abolish its perceptibility to the hand, that is, by copious pleuritic exudation, pneumothorax, and to a moderate degree also by the presence of a large quantity of fluid in the air-passages (see p. 64 *et seq.*)

INCREASE IN THE INTENSITY OF THE VOICE. BRONCHOPHONY.

Bronchophony, like bronchial respiration, occurs as a *physiological* phenomenon, in the larynx and opposite the bifurcation of the trachea; but at the latter level it has already lost much of its force, and is heard only to the right of the vertebral column.

Pathologically, bronchophony may occur at any part of the thorax; but other conditions being equal, it is louder superiorly, both in front and behind (in the interscapular space), than it is inferiorly. In its more exaggerated forms its recognition is easy without the institution of any comparison with the healthy sounds; when its special characters, however, are less markedly developed, such a comparison is necessary, with the vocal resonance at the corresponding points on the sound side of the chest, or indeed with that at any part of the lung which may have preserved its normal texture.

Bronchophony is invariably an indication that in the region auscultated the lung is somewhat extensively *consolidated* or *contains a large cavity enclosed by solid walls.*

The expression "consolidation of the lungs" embraces a well-known class of pathological processes, amongst which the hepatization of pneumonia and caseous condensation (phthisical) are found to be the commonest causes of bronchophony.—This intensification of the voice-sounds is observed more often in phthisical than in bronchiectatic vomicæ, the former being more favourably situated for its development; it is but seldom, also, that gangrenous cavities attain sufficient size to give rise to bronchophony.—In very large phthisical excavations *bronchophony* has a *metallic*, ringing character, exactly as in the case of the respiratory murmur and the cough (see p. 164). This ringing sound, however, is not so loud as that which accompanies the cough, as the latter throws the air within the cavity into more powerful vibration; phthisical patients presenting caverns in the lungs, also, are usually greatly reduced in physical strength, the voice, therefore, is weak, and is further frequently rendered husky from concurrent laryngeal affection.

Bronchophony has, with regard to the determination of the physical condition of the lung, exactly the same diagnostic signification as the bronchial respiratory murmur, and in all instances its occurrence, its distinctness, its temporary disappearance and its reappearance, are dependent on precisely the same circumstances as in the case of bronchial respiration; this is true more particularly with reference to the size of the condensed part or of the cavern, which must be at least so large as to include one of the more important bronchi, and to the free communication which must subsist between this bronchus and the trachea (see p. 66).

Since bronchial respiration and bronchophony arise from the same causes they are always found associated, though not invariably of equal intensity. The presence of large accumulations of fluid in the condensed portions of the lung or in cavities, irregular infiltration of the alveoli, or the existence of spongy lung-substance between patches of consolidation, diminish the clearness of bronchophony, or prevent its development altogether.—Like bronchial respiration, it is always combined with increased pectoral fremitus,—a fact the reasons for which are self-evident; but much finer shades of difference may be detected by auscultation than by palpation, and when, moreover, bronchophony is confined to a very circumscribed area, examination by the latter method gives very unreliable results. It is customary, therefore, except in cases of copious pleuritic effusion and other affections which render large parts of the lung void of air, to investigate the anomalies of the voice by auscultation rather than by palpation.

Læennec drew a sharp line of demarcation between two forms of increase in the intensity of the voice,—pectoriloquy and bronchophony. By the first term he designated the loud and exaggerated quality of voice heard in very large and superficial pulmonary caverns. But pectoriloquy is in no essential respect different from bronchophony; it is simply the same character of voice in its highest degree of intensity. If so disposed, therefore, one may, with Skoda, distinguish between a faint and a loud bronchophony, the latter being merely Læennec's pectoriloquy, though these slight differences have no particular diagnostic meaning, just as there is no special physical condition corresponding to loud and feeble bronchial respiration.—Bronchophony is generally loudest in cavities; but this is not the invariable rule, as it is occasionally observed to be weaker in such circumstances than in tissue which is simply infiltrated, but free from excavation.

Bronchophony is also heard when the lung is compressed by pleuritic exudation, *but only at those parts of the chest at which the dense lung is in immediate contact with the chest-wall*, that is, *posteriorly*, between the vertebral column and the scapula, when the effusion is non-encysted.—One condition which seems to be essential to the development of bronchophony is that the larger bronchi must still be patent, to permit the entrance of the waves of sound; should these too be closed by reason of the greatness of the pressure bronchophony is wanting. At those parts, on the other hand, at which the lung is separated from the chest-wall by the effusion, the vocal resonance, like the pectoral fremitus, is feebler than on the sound side. Nevertheless, all exudations do not offer the same opposition to the transmission of the voice-sounds; Baccelli has shown that the more fluid and homogeneous the effusion the more easily and completely is it penetrated by the vocal vibrations, and that in favourable circumstances even a whisper may be audible through effusion; but through fibrinous, and particularly through purulent exudations the waves of sound pass with difficulty or not at all. There is thus a possibility of ascertaining, by auscultation of the voice, especially of the whispered voice, whether the exudation is serous, fibrinous, or purulent. To facilitate the appreciation of these differences in the intensity with which the voice is conducted Baccelli recommends that the auscultating ear should be pressed firmly against the spot under examination, that the other ear should be closed with the point of the finger, and that when the patient speaks, either aloud or in a whisper, he should turn his face away from the head of the examiner.

Compression of the lung from other causes than the presence of fluid is seldom so extensive or so complete as to offer conditions favourable to the production of bronchophony; for the same reasons, also, pneumothorax is not characterised in every case by bronchophony, and in those instances in which it does occur it is heard only posteriorly, where the collapsed lung lies close to the spinal column, whilst anteriorly and laterally the voice is weakened or suppressed.

Physical Cause of Bronchophony.

The *cause* of Bronchophony is to be found in the superior sound-conducting power of *impermeable* lung-tissue. This increased vocal resonance is never observed in the thorax in health, as spongy expansible lung is composed of non-homogeneous (dense and rare) media, namely, air and pulmonary substance, or the tissues forming the walls of the bronchi; the vocal sounds are thus constantly refracted, and therefore weakened. But should these unfavourable conditions be removed by pathological processes which render a large part of the lung more or less solid, and so change it into a body of uniform consistence, the development of bronchophony is certain.—This theory, which was first started by Læennec, and which has already been described in detail when treating of the causes of bronchial respiration, explains in a perfectly satisfactory manner all the phenomena of bronchophony.

In accounting for both bronchial respiration (see p. 136) and bronchophony Skoda combats the idea that either of the phenomena arises from the better conduction of the voice-sounds through the dense pulmonary parenchyma; the incorrectness of his views on this subject, however, has been shown on the above-mentioned page. This, nevertheless, seems to be the most fitting place in which to discuss fully the theory of *consonance*, which Skoda brings forward as the cause of bronchophony in all cases in which it occurs. He developes his theory in the following way:—

When the voice as heard in the thorax has the same intensity as at the point at which it is formed (the larynx), we have to do with a condition which must obviously have its origin in one of two ways: either the voice is concentrated whilst being propagated from the larynx to the ear of the observer,—the principle on which the speaking-trumpet is constructed, or it is reproduced and intensified by consonance.

Should the voice be *louder* in the thorax than over the larynx the conclusion that this change is the result of consonance is *unavoidable.*—The voice as it issues from the mouth is augmented in volume and force by consonance, the shock of the original sound generated in the larynx throwing the air in the buccal and nasal cavities into simultaneous vibration; closure of the mouth and nose, also, produces an immediate alteration in the timbre of the voice. The vocal sounds must further consonate in the air of the trachea and bronchi in the same way as in the air-spaces just mentioned (the pharynx, mouth, and nose); this is clearly indicated by the pulsation of the trachea in speaking. But this vibration is not transmitted directly through the walls of the larynx and trachea to the bronchi, this being prevented by the dissimilar structure of these parts, otherwise bronchophony should be heard over the whole of the thorax; the voice can therefore consonate only in the *air* of the bronchi, and that only when this air is pent up in an enclosed space, as in the sounding-board of a stringed instrument. In *normal* circumstances, however, the bronchi constitute but very imperfectly enclosed spaces, their parietes being composed of non-homogeneous tissues; thus, whilst the trachea and its two primary divisions are tubes of a perfectly uniform cartilaginous structure, forming chambers well adapted for the manifestation of consonance, the bronchi immediately beyond are not surrounded on all sides by complete rings, their walls being strengthened only by small cartilaginous plates, which become fewer and thinner the further the ramifications of the bronchi are followed out, and finally disappear entirely in the bronchioles. The voice in these bronchi, therefore, becomes progressively weaker and is ultimately almost lost to the ear, its sonorous waves being broken up and dispersed through the substance of the lung and the thoracic parietes. But if the walls of the bronchi become more solid, and so acquire a uniform structure, by morbid processes which condense the pulmonary parenchyma and render it void of air, the air-passages are transformed into closed spaces, surrounded by dense rigid parietes, and present then the most favourable conditions for the development of consonance of the voice; that is, the air within them is caused to vibrate by the voice, and the vibrations so aroused are reflected by the firm walls and go to add to the force of the originally feeble waves of sound proceeding from the larynx. Intensification of the voice in this manner furnishes at least *one* explanation of the fact that it is occasionally audible over the thorax even in the presence of conditions which hinder its free transmission to the surface; but it seems probable also that these reinforced voice-waves excite the surrounding airless dense tissue to simultaneous vibration, exactly as in the case of the walls of the larynx, so that the voice reaches the ear of the auscultator *unimpaired in intensity.*

In the first place it must be stated that the circumstance on which this theory is chiefly founded,—that the voice in the *thorax* is, under certain pathological conditions, louder than over the *larynx,*—is one of exceeding rarity, and one the existence of which seems to me, in common with many other observers, to be open to doubt; but even if such cases do occur they offer no conclusive proof that the voice as generated in the larynx is *weaker* than that heard over a pulmonary cavern or consolidated lung-tissue, as in auscultating over the larynx only a portion of the laryngeal voice-waves which pass in a downward direction in the air-passages is collected in the tube of the stethoscope, the latter being placed perpendicularly on the surface,—an unfavourable position for receiving the whole of the sound. To ascertain the real strength of the laryngeal voice it is necessary to auscultate in the mouth, and here the voice is found to have such a degree of intensity as it never has over the thorax. And Skoda's argument that the same obstacles are encountered in the propagation of the voice through the thoracic parietes as through the wall of the larynx, is inapplicable to very large and superficial caverns,—and it is only in such cavities that very loud bronchophony is heard,—as in these the waves of sound reach the ear undiminished in intensity, being transmitted in the direction of the periphery of the lung, that is, exactly in the direction of the stethoscope (Wintrich).—Further, the assertion that the vibration of the larynx in speaking is communicated only to the column of air enclosed by the bronchi, is incorrect; the walls of the bronchi also are caused to vibrate.—Apart from these considerations, on purely physical grounds, the theory of consonance is rejected by many authorities (Wintrich, Schweigger, &c.) as untenable as an explanation of the causation of bronchophony in most of the cases in which it is observed. Thus, in enclosed spaces the air can consonate only with tones whose sound-waves are of equal length, and therefore of equal height, that is, usually with only *one* tone, the fundamental tone; and further when this fundamental tone is characterised by a certain degree of intensity consonance may take place with those sounds to which it bears a harmonic relation (1 : 2 : 3 : 4, &c., that is, its octave, its twelfth, its double octave, &c.)—in other words, with its *higher harmonic sounds.*

Skoda is also of opinion that the air-containing spaces in the lungs cannot be properly compared with those formed by closed tubes, which are capable of entering into consonance only with certain sounds; he holds that the bronchi, or pulmonary excavations, are rather "air-spaces of very varied form and structure, differing widely as regards their mode of communication with the larynx, so that the number of their consonating notes must of necessity be large, and that the compass of the voice in speaking is not so great that enclosed spaces of so exceedingly diverse conformation are unable to supply the appropriate harmonic sounds."—This, however, is a somewhat forced argument; if such spaces, adapted to the production of consonance with any tone whatever, really existed in the lungs, the thoracic voice at parts corresponding to the situation of pulmonary cavities should, even in speaking, and still more when tones of different pitch are sounded, show great differences in intensity according as the original tone is or is not in unison with the proper tone of the cavity. But this is not usually the case; it is only in very large excavations, (but never in simple consolidation of the lung), that signs are observed which point unquestionably to the presence of consonance of the voice in the lungs. Such an indication is the formerly-mentioned (p. 170) ringing metallic quality acquired by the voice-sounds, amphorophony, heard when single words are spoken in a tone of a certain pitch. The applicability of Skoda's consonance-theory is therefore limited to the so-called metallic (amphoric) phenomena which may accompany the voice, the cough, the respiratory murmur, and râles (see pp. 139 and 156).

ÆGOPHONY.

This term is employed to designate a peculiar tremulous, interrupted quality of voice, which in timbre resembles the bleating of a goat, the nasal twang of the human voice when the nostrils are closed, or the sound produced by speaking against a comb which is covered with paper and held in front of the teeth; it very frequently creates the impression also as if it came from a great distance. It is very commonly met with in cases of moderate pleuritic exudation, (not if the effusion be exceedingly abundant or of very small amount), and usually towards the upper margin of the fluid; it is most often heard in the space included between the axillary line, the inferior angle of the scapula, and the vertebral column. All the sounds uttered by the patient do not assume the bleating character, and those which do so have not all the same timbre. When such a change, from the bleating to the non-bleating voice, takes place in pleurisy, the former is higher in pitch and seems remote from the ear, the latter deeper and closer to the ear.

Ægophony lasts, in some cases, for a considerable length of time, when the condition which gave rise to it, the presence of exactly the requisite amount of fluid in the pleura, undergoes no change; but it quickly disappears on increase of the exudation.

Ægophony is most probably produced by the vibration of the walls of the flattened, compressed bronchi; this vibration is excited by the voice, and transmitted to the thin layer of fluid which, at the upper part of the exudation, lies between the lung and the chest-wall. This tremulous movement of the sides of the bronchi gives the voice-sounds a quavering, interrupted character; and as they have to pass through a fluid medium to reach the surface they lose in clearness and precision, and acquire the nasal twang.

The diagnostic signification of ægophony, so far as it reveals the physical condition of the pulmonary substance in pleurisy with exudation, is the same as that of bronchophony, of which, indeed, it is simply a modification. It is not unusual to find ægophony and ordinary bronchophony in the same patient, and at points not far distant from each other.

According to Skoda ægophony occurs not only in cases in which there is fluid in the pleura, but also in the absence of such fluid, in some cases of pneumonia, in caseous pneumonic infiltration of the lung substance, with or without excavation, and sometimes even between the shoulder-blades of children in perfect health; no satisfactory explanation has yet been offered of its causation in such circumstances.

EXAMINATION OF THE SPUTA.

Almost all diseases of the organs of respiration are accompanied by more or less considerable catarrh of the bronchial mucous membrane, the secretions resulting from which are expelled by *coughing*. This coughing is characteristic of the expectoration of fluids secreted by the lining membrane of the respiratory apparatus proper, those from the pharynx or nares being got rid of by a quick, hawking expiration. Expectoration may also be entirely wanting at various stages, or during the whole course, of the pulmonary affection; its absence therefore does not *exclude*, nor does its presence *prove*, the existence of disease of the respiratory organs. Still another source of error has to be guarded against: the secretions may pass downwards from the fauces or nose into the larynx, and are then naturally discharged by coughing.

The sputum in the different diseases of the lungs consists of very diverse morphological and amorphous elements, by which its appearance and general characters are so modified that a rough estimate of its composition may usually be made even on examination with the naked eye, though its exact constitution can be determined only by means of the microscope.

Morphological Elements of the Sputa.

1. *Epithelium.* The expectoration usually contains pavement epithelium, more seldom columnar epithelium, most rarely of all ciliated epithelium.

The *pavement epithelium* may be derived from the upper portion of the air-passages; it usually comes from the buccal mucous membrane, however, and is mechanically mixed with the sputum in the passage of the latter through the mouth.

It is easily recognised by the polygonal outline of the individual cells, by their size, and their large nucleus.

Epithelium of this kind is most abundant in the sputa of those suffering from catarrh of the pharynx and mouth. The epithelial cells of the deeper layers of the buccal mucous membrane are more flattened, have a very indistinct nucleus or none at all, resemble more the cells of the epidermis, and are in no way distinguishable from the pavement epithelium of the upper part of the air-passages.

Columnar epithelium is somewhat rare as a constituent of the sputum, though the air-passages are lined with it from the larynx down to the finest bronchioles. Its attachment to the mucous surface is of the most intimate nature, severe catarrhal swelling of the membrane, or even destructive morbid processes, being insufficient to separate it in any quantity. The *epithelium of the pulmonary alveoli* occasionally, though rarely, appears in the sputum.

Ciliated columnar epithelium is very seldom present in the expectoration in diseases of the air-passages; it is found oftener in catarrhal secretions coming from the mucous membrane of the nose and the sinuses connected with it.

2. *Pus corpuscles* (colourless blood-corpuscles).—These are simply the white blood-corpuscles, which pass through the walls of the capillaries in inflammation (Cohnheim); they occur in variable proportion, according to the intensity of the catarrh or inflammatory process, and are sometimes so abundant as to form the sole representatives of the morphological element in the expectorated matters. The sputum acquires certain macroscopic characters, which vary with the amount of pus present; it is thus rendered more or less opaque, and yellow or yellowish-green in colour.

Besides pus corpuscles there may usually be recognised in the sputa *mucus* corpuscles, shrunken or fatty cells, cell-nuclei, and granular masses.

3. *Red blood-corpuscles.*—Blood is expectorated sometimes merely in traces, at other times in more perceptible quantity, and occasionally so abundantly that it seems free of any intermixture with other matters, and itself to constitute the whole of the sputum. The presence of even the smallest trace of blood is generally indicated by its colour; but if the tinge be so slight as to leave any doubt, the question may be easily settled by microscopic examination.

Blood-corpuscles in the sputum exhibit their normal histological form and colour, as they are placed in precisely similar conditions as while still in circulation, that is, they are in a fluid of alkaline reaction and containing alkaline salts. No distension or discoloration of the corpuscles takes place, therefore, from absorption of water; it is only when the quantity of blood present is very small, and the proportion of water consequently large, that the phenomena of diffusion are observed.

4. *Débris of lung-tissue.—Elastic fibres.* These are met with in the sputum only in those diseases which involve destruction of the tissue of the lungs and bronchi,—caseous pneumonia, bronchiectasis with ulceration of the bronchi, and pulmonary abscess. The portions of sputum in which they are suspended may often be distinguished, by their dull grey opaque colour, from the clearer mass of the expectoration. On being allowed to stand for a short time in a vessel they sink to the bottom with the heavier constituents, which are generally free of air-bubbles. The elastic fibres appear either as isolated, detached filaments, or collected into bundles, and these may be either straight or coiled and twisted; occasionally, also, they are interlaced after the fashion of trellis-work, or are so perfectly meshed as to closely resemble a net, and in the latter case present exactly the appearance of the fibrous framework of the alveoli. They are brought markedly into prominence by their dark colour when under a powerful light, particularly on the addition of acetic acid, which renders the rest of the tissues more transparent but has no effect on the elastic fibres.

The presence of elastic fibres may generally be regarded as diagnostic of phthisical disorganisation of the lungs, phthisis being the most common of the pulmonary diseases leading to destruction of tissue. Lung-tissue, however, occurs also in the sputum in cases of pulmonary abscess, of ulceration of the bronchi, and of bronchiectasis, but *not*, or only for a short time and in small quantity, in cases of gangrene of the lungs. In the last-named affection the elastic fibres are destroyed by the action of some imperfectly known substance, which is apparently of the nature of a ferment (see p. 203).

The discovery of these fibres in the expectorated matters in phthisis gives no indication as to the exact seat of the morbid process; they may come either from the walls of the alveoli or from those of the finer bronchi. The distinction which was formerly made, according to which the long, straight or slightly coiled fibres were supposed to

6. *Crystals.*—Those which occur most frequently in the sputum are the *crystals of the fatty acids,* which, though formerly thought to be simply margaric acid, are now known to consist of a combination of palmitic and stearic acids. Under the microscope, with a magnifying power of 300 diameters, they have the appearance of long, slender, colourless needles, lanceolate in shape, usually straight, sometimes curved, and occasionally also somewhat varicose. They are sometimes observed singly, detached from each other, at other times grouped in tufts or sheaves. In the sputum they are always found adhering to shreds or masses of matter of a dull greyish colour, and of exceedingly offensive odour. These shreds are met with in the expectoration which comes from gangrenous or bronchiectatic cavities, and in that of putrid bronchitis. The presence of crystals in the sputum is thus indicative of some morbid process involving destruction of tissue; they are observed, similarly, in gangrenous discharges from other parts of the body. They were first demonstrated by Virchow.—Their microscopic appearance is so characteristic that they can scarcely be mistaken for anything else; when the needles happen to be curved, however, they resemble somewhat closely the elastic fibres formerly referred to. In the latter case their chemical reaction serves to distinguish them at once; on the addition of chloroform or ether, the needles are quickly and completely dissolved, while the elastic fibres undergo no alteration.

Another kind of microscopic *crystals*, having the form of elongated, very pointed octohedra, of rhombic plates, or of fine, sharp, spindle-shaped bodies, has been found in the sputum in various diseases. They are colourless, the largest 0·01—0·02 mm. long, the smallest visible only under a manifying power of 500 diameters. Their chemical nature is still obscure, though they are supposed to be of organic origin. They are insoluble in alcohol, and may therefore be preserved in spirit; but concentrated acids and alkalies dissolve them rapidly. They resist also for a long period the disintegrating action of putrefaction. These crystals, which were first noticed by Charcot, and with which his name is now usually associated, occur very frequently in leukæmia,—in the spleen, the blood, and the marrow of the bones. They may also be seen, and apparently not unfrequently, in the sputum in various affections of the bronchi, in chronic bronchial catarrh, in pulmonary emphysema, in the expectorated casts in bronchial croup, but relatively most often in bronchial asthma, (as in six out of seven cases investigated by Leyden) particularly during, and immediately after the attacks, not usually in the free intervals.

Leyden thinks it not improbable that these crystals are the direct causes of the dyspnœal seizure in bronchial asthma, his hypothesis being that they irritate the terminations of the vagus in the mucous membrane of the air-passages, and in that way, by reflex action, give rise to spasm of the finer bronchi; the remissions would thus follow the expectoration of the crystals, and the patient would have ease till the renewed formation of crystals produced another attack. But in a great many cases, in which there is no preceding dyspnœal seizure, such crystals are found in the sputum, as Zenker observed in his own case, and in two instances of bronchial croup, in which the crystalline masses were incorporated in the fibrinous casts, embedded chiefly in their periphery.

Other crystalline formations also are occasionally seen in the sputum: *hæmatoidin*, for instance, ocurring as rhombic rods or as slender needles arranged in tufts, in cases of bronchiectasis, of empyema and peritoneal or hepatic abscesses which have opened into the bronchi; crystals of *cholesterin*, in cases of pulmonary abscess or empyema penetrating the lung, and sometimes in the phthisical sputum; *tyrosin*, also, is met with in the expectoration in putrid bronchitis.

7. *Fungous growths.*—The parasitic fungi which occur in the sputum are the thalli and spores of the *Leptothrix buccalis* and the thrush-parasite (*Oidium albicans*), and in rare cases *Sarcinæ.*

When the buccal cavity is lined with fungous growths, such as the more common and widely-distributed oidium albicans or the leptothrix buccalis, these parasitic vegetations frequently appear in the sputum, being detached from the mucous membrane and mixed with the expectoration in its passage through the mouth; in these cases their quantity is not great.—But such minute vegetations, found in the sputum, may come also from the lungs: they are sometimes transplanted from the mouth to the lungs, being carried downwards by the inspiratory current of air; there they grow rapidly, finding in the stagnant secretions of dilated bronchi and pulmonary cavities a soil which favours their development.—Leptothrix and oidium albicans are observed in the sputum in putrid bronchitis and gangrene of the lungs. It is not improbable that the offensive decomposition of the bronchial and cavernous secretions which is so characteristic of these affections is due to the presence of fungi or of the bacteria which gain entrance with them. It appears also that even in healthy respiratory organs, under certain conditions, the introduction of microscopic fungi is sufficient to excite a kind of putrefactive action leading to putrid bronchitis (Rosenstein).

Large numbers of fungous growths, both thalli and spores, have been detected in the sputum in hooping-cough (Letzerich).

On treating such a sputum with iodine and concentrated sulphuric acid the filaments assume a beautiful blue colour, while the spores become brown.

Letzerich regards these bodies as the cause of hooping-cough (?); the frequency of the spasmodic seizures, according to him, depends on the greater or less rapidity of the development of the vegetations and on the irritability of the affected part of the mucous membrane, while the disease becomes less severe as soon as an abundant secretion of mucus takes place, along with which the parasites are expectorated. These fungi, taken from the sputum of a patient suffering from hooping-cough and introduced (through a fistula) into the trachea of a rabbit, produced in the latter catarrhal symptoms and a paroxysmal cough, and the sputum discharged contained growths exactly similar to those found in the original disease in the human subject.

Multitudes of *vibriones* are also frequently observed in the secretion of bronchiectatic and gangrenous cavities, and in that associated with various other disorders. When discovered in large quantity in sputum recently coughed up they probably come from the respiratory passages; they are found, however, in every sputum which has been exposed some time to the air. Occasionally, also, they are mechanically mixed with the expectoration in the mouth, being abundantly developed in the tartar which loads the teeth of those who do not regularly cleanse the mouth.

In a few cases of phthisis (recorded by Virchow, Cohnheim, and Heimer in Ziemssen's clinic) *Sarcinæ* have been seen in the sputum and in the lungs in considerable numbers, while at the same time no trace of their presence in the stomach was discernible; they were found in none of the other organs and were of very small size.

Echinococcus-vesicles, whole or in fragments, occur equally rarely in the sputum. When present they may proceed either from the liver, when a hepatic hydatid cyst, making its way through the diaphragm into the lung, perforates a bronchus, or from the lung itself, when the parasite is primarily developed in that organ.—There are more than 40 well-authenticated cases in which hydatid vesicles were expectorated; in only one out of fourteen cases of hydatids that have come under my own observation was this symptom present.

Particles of food, muscular fibres, grains of starch, vegetable fibres, &c., which may all be *accidentally* mixed with the constituents of the sputum, are so readily distinguished by microscopic examination that a particular description of them is unnecessary.

AMORPHOUS ELEMENTS OF THE SPUTUM.

These constitute the basis of the sputum, and consist chiefly of mucus, albumen, and the watery fluids by which the mouth is kept moist.

Mucus (Mucin) is the proper secretion of the bronchial mucous membrane, and is present in every sputum. It is of varying consistence, being sometimes fluid, and at other times more viscous and capable of being drawn into threads; to the naked eye it is transparent. The chemical test by which it is most commonly distinguished is the formation of a cloud or precipitate on the addition of acetic acid.—The presence of mucus in the sputum has absolutely no diagnostic significance; persons in perfect health expectorate more or less of it every day, and in all forms of lung disease, from the mildest catarrh to the most severe affections, it is a constant constituent of the secretion.

Water, also, forms a considerable part of all sputa. The more abundant it is the more fluid is the expectoration. The water is commonly supplied by the mucous lining of the mouth, and in that case contains also some pavement epithelium; in certain other cases it comes from the air-passages, (when the bronchi are flooded with a profuse secretion of serous fluid), or from the pulmonary aveoli, (as in œdema of the lungs). The sputum in bronchial catarrh is at first thin and aqueous, but subsequently becomes more tenacious and deficient in water.

Albumen occasionally enters into the composition of the sputum, and it is always more abundant the severer the inflammation of the air-passages or pulmonary substance. It is present in greatest quantity in cases in which there is plastic exudation into the parenchyma of the lung and into the finer bronchi,—that is, in pneumonia.—Albumen is detected by acidulating the alkaline sputum with acetic acid, filtering, and boiling, or by adding nitric acid: in both cases the albumen is coagulated.

General Classification of Sputa.

Biermer arranges the sputa into the following groups, according to the nature of their principal constituent and the predominance of one or other microscopic element, conditions which are usually more or less easily distinguished even with the naked eye.

1. *The mucous sputum.* This consists almost exclusively of mucus, and is frequently brought up by perfectly healthy individuals, and in the earlier stages of bronchial catarrh.

2. *The muco-purulent sputum*, containing mucus and pus-cells. The latter constitute the sediment which falls to the bottom of the crachoir when the sputum is allowed to stand a short time, whilst the mucus and the air-bubbles usually scattered through it occupy the upper part of the vessel, floating on the top of the water. The miscibility of these sputa with each other depends on their consistence; they either run together, forming a thin, uniform fluid, or fail to coalesce, being divided into numerous viscid, globular masses. They occur both in simple bronchial catarrh and in every other affection of the bronchi and of the substance of the lungs, and are consequently the most common variety of sputum.

3. *The purulent sputum* is homogeneous in character, has exactly the colour of pus obtained from an abscess, is thick, though not capable of being drawn into threads, and sinks to the bottom of the expectoration-glass. Microscopically it is seen to consist almost entirely of pus-cells.—It is derived from suppurating pulmonary vomicæ, or from a pleural cavity filled with pus, which perforates and discharges through a bronchus (empyema).—A purely purulent sputum is somewhat rare.

All sputa, may, with more or less justice, be included in these three groups, as all contain mucus or pus. But when, in certain circumstances, other constituents come into decidedly greater prominence, in respect either of their quantity or their morbid significance, the term by which the sputum is designated is determined by these; a good example of this is found in the *sanguineous sputum.*

This classification of the sputa into mucous, muco-purulent, purulent, and sanguineous, has this practical advantage, that the element most characteristic of the secretion in the individual case is at once and plainly expressed. It is obvious, however, that it is only approximately, and in a small proportion of the cases, that the origin of the particular sputum and the nature of the disease which gives rise to it are indicated by these terms. A strictly accurate estimate of the nature and diagnostic importance of the sputa is possible only on taking into consideration a number of other signs, which are now about to be discussed: these are the form, consistence, weight, colour, odour of the sputum, and very specially the presence of the formerly-mentioned histological elements (fragments of lung-tissue and pathological products).

PHYSICAL CHARACTERS OF THE SPUTA.

Consistence of the sputum. This varies within very wide limits, all intermediate conditions being met with, from a consistence almost watery to that presenting the utmost degree of viscosity. The tougher the sputum the firmer is its consistence; the mucous and muco-purulent secretions, therefore, attendant on acute bronchial catarrh and the parenchymatous diseases of the lungs, (the cavernous sputa, for example), possess this character in a high degree.—If the sputum be deficient in mucus, the substance which binds together the various histological elements, it becomes less coherent; the simply purulent sputum is thus much less tenacious than the muco-purulent form.—Persistent expectoration of very viscid sputum points to a state of intense irritation of the bronchial mucous membrane.

The *form of the sputum* depends on its consistence. Very fluid sputa, such as the serous expectoration of pulmonary œdema or the purulent discharge from a pulmonary abscess or an empyema which has perforated one of the larger bronchi, do not remain separate but become intimately blended with each other in the crachoir, forming a homogeneous mass; very tough sputa, on the other hand, are irregularly globular in form, while less tenacious secretions become somewhat flattened or nummular. Mucous and muco-purulent sputa sometimes coalesce, and at other times assume various shapes determined by the greater or less consistence of the mucus. The spherical or nummular form prevails in the muco-purulent sputa proceeding from phthisical cavities, the configuration of each sputum being the more distinctly preserved the less the quantity of fluid bronchial secretion subsequently mixed with it. If the latter be very abundant, the circular, coin-shaped sputa, at first quite distinct from the catarrhal secretion, unite with and are ultimately uniformly diffused through the homogeneous layer of mucus after standing some time in the vessel.

Weight of the sputa. The denser the sputum, and the fewer the air-bubbles it contains, the greater its weight. Sputa, therefore, of firm consistence and free of air sink to the bottom of the glass, while those of more fluid consistence and full of air-bubbles float about at the top. Sputa which sink in water preserve their form (cavernous sputa); the other less dense varieties soon break up into several layers, the lighter elements, air and mucus, swimming on the surface of the water, the heavier constituents, such as pus-cells, subsiding and forming a sediment at the bottom. The weight of the sputum thus indicates only its consistence, and usually also the elements which enter into its composition, but gives no clue as to its origin; the expectoration observed in bronchial catarrh in the stage of resolution may be just as heavy as that secreted by a pulmonary cavern.

The *quantity of the sputum* is exceedingly variable, in the acute as well as in the chronic diseases of the respiratory apparatus. Occasionally in the course of acute affections there is absolutely no sputum, and in chronic disorders also it may be wanting for a considerable period. In the severest types of disease the quantity of matter expectorated may be very small, while in much less grave affections it may be extremely great. General rules, therefore, with regard to the prognostic value of increase or decrease in the amount of the sputum in the different diseases of the respiratory organs cannot be laid down. Nevertheless, the following may be accepted as a generalisation, so far as this is possible, of what is known on the subject: in acute bronchitis, hooping-cough, and pneumonia the expectoration becomes more abundant,—and is then often a critical indication of the approaching termination of the disease,—simultaneously with the disappearance of the difficulty with which it is brought up, and when it begins to assume more definite shape and consistence; each exacerbation of the affection, on the other hand, materially diminishes the amount of the previously profuse secretion, while the difficulty and pain of expectoration increase, and the sputum acquires greater density. When, in acute diseases (bronchitis and pneumonia, for instance), the expectoration becomes scantier or fails altogether, while at the same time the morbid process is obviously extending and increasing in severity, and while auscultation shows that the bronchi are loaded with an accumulation of fluid, we have direct evidence of a lowering of the irritability of the sensory terminations of the vagus in the lungs, or of the profound exhaustion of the patient; this sign is therefore one of grave import. The stertorous respiration of the dying, accompanied by rattling sounds produced in the chest and audible at some distance, furnishes the most familiar example of this condition.

It is in *Bronchiectasis*, of all diseases of the respiratory apparatus, that the largest quantity of expectoration is discharged at one time. This fact, in the absence of such other signs as would establish the diagnosis between this and like conditions, becomes a criterion of the first importance. The expulsion of several tablespoonfuls, and often more, of a muco-purulent, fœtid secretion, as the result of one effort of coughing, is far from uncommon in this affection.—In like manner the *bursting of a pulmonary abscess*, or of a purulent *pleural exudation*, into one of the larger bronchi, is marked by the sudden evacuation of a large quantity of purulent, homogeneous sputum.

Odour of the sputum. This is quite wanting in very many cases, or is at most faint and of a somewhat mawkish or mouldy character. The sputa in pulmonary abscess, bronchiectasis, and putrid bronchitis, have a decidedly fœtid odour, that of the discharge in gangrene of the lungs being extremely offensive. In the latter case the expired air is already considerably tainted even before any trace of a disagreeable smell is perceptible in the sputa. The fœtor is caused by the gangrenous putrefaction and disorganization of the tissues.

Putrefying fragments of food, such as those which adhere to the teeth, may communicate this offensive odour to the sputum in its passage through the mouth. Caries of the teeth, and especially certain affections of the mouth, are apt to produce the same result; it is from this source that, in the last stages of phthisis, in which various disorders of the cavity of the mouth, associated with the development of fungous growths, are so common, the sputa derive their odour of putrescence. This is lost when the expectoration has stood some time in the vessel.

COLOUR OF THE SPUTA.

A slight coloration, varying from white to yellow or yellowish-green, is given to the sputum by the presence of a large number of pus-cells. A deeper tint is usually due to the admixture of the colouring-matter of various tissues; these are, enumerated in the order indicated by the frequency of their occurrence, the red colouring-matter of the blood and its different modifications, the biliary colouring-matter, and a black pigment.

RED, SANGUINEOUS SPUTA.

These consist either of blood alone, or of blood and other elements intermixed.

1. A sputum composed *solely* of blood, and expectorated in any considerable quantity, is invariably the result of rupture of some of the vessels of the lungs. The amount of blood thus lost at one time is very variable; it may be only a teaspoonful, or as much as several tablespoonfuls, or even more.

Hæmoptysis frequently occurs at the outset of pulmonary phthisis, at a time when the physical signs of the affection are still undeveloped; or it may take place, either once or repeatedly, in the course of the disease. Hæmoptysis may also be caused by affections of the cardiac valves, when these lead to overloading of the pulmonary circulation; an example of this is seen in *hæmorrhagic infarction* of the lungs following lesions of the mitral valve.

Blood effused into the lungs is for the most part *coughed* up immediately after the hæmorrhage.*

This fact affords one of the readiest means of distinguishing pulmonary hæmorrhage from hæmatemesis (in cases of round ulcer of the stomach, carcinoma, &c.), the blood in the latter case being rejected by *vomiting*. The *bright red* colour, the *fluid condition*, and *frothy* appearance of blood proceeding from the lungs, are further points which aid in establishing the diagnosis, blood coming from the stomach being usually dark reddish brown or chocolate-brown in colour, having a close resemblance to *coffee-grounds*, and sometimes, when it has remained some little time in the stomach, forming clots in which are embedded fragments of food.—The mere external appearance of the blood, however, does not always furnish sufficient data on which to found an opinion regarding its origin. On the one hand, blood poured into the lungs may be retained there for some time, lodging particularly in dilated bronchi or in pulmonary cavities, when it becomes much darker in colour and is slightly coagulated, being thus not unlike that discharged in hæmatemesis; blood effused into the stomach, on the other hand, may be vomited immediately after escaping from the ruptured gastric vessels, when it has no longer the appearance above described, but resembles rather blood coming from the lungs. And finally, blood flowing from the nose, mouth, or pharynx, may, during sleep, pass through the larynx and into the bronchi, when it takes on a brighter red colour and receives a certain admixture of air, and as it is eventually brought up by coughing it may possess all the characters of blood originally extravasated in the lungs. In such cases the source of the hæmorrhage can be determined only on consideration of the patient's previous history and of the results of careful physical examination.

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* Whether the blood remaining in the alveoli and ultimate bronchi after pulmonary hæmorrhage, when not entirely absorbed, excites inflammation and becomes the starting-point of caseous infiltration (F. Niemeyer), is still a disputed question. It has been clinically proved (Traube) that after the lapse of a certain time no trace of bleeding is discoverable in the lungs. Experiments on animals give different results according as the blood injected penetrates only to the finest bronchi or reaches the alveoli. Should the blood not go beyond the air-tubes its absorption is completed within twelve hours, and no inflammation follows (Perl and Lipmann); if it be forced into the alveoli, however, catarrhal pneumonia is set up, which, in healthy animals, ends in resolution (Sommerbrodt), but in men placed under unfavourable conditions (men of feeble constitution, &c.), results in caseous infiltration and phthisis. That hæmorrhagic infarctions in cases of cardiac disease never give rise to catarrhal pneumonia is probably in some way connected with the immunity from phthisis which the subjects of diseases of the heart seem to enjoy.

2. Blood may be *mingled* with the sputum in various proportions, intimately, or in streaks, or simply dotted through the mass. In these cases the presence of the blood need not necessarily be assumed to be due to rupture of any of the pulmonary vessels; it may be caused by the passage of red blood-corpuscles through the uninjured walls of the vessels (bleeding *per diapedesin;* Cohnheim).

The depth of the coloration of the sputa is in proportion to the quantity of blood present; whatever be the amount the fundamental colour is always red.

The lighter shades of red are produced by the greater or less predominance of the other constituents of the sputum; they depend also, to a certain extent, on the physical characters of the expectoration, varying according as the latter is of fluid or firm consistence, mucous or purulent, or has become tainted by putrefaction, &c.

The blood is the more *intimately* mingled with the other elements of the secretion the longer it has been in contact with them; the more tenacious the bronchial mucus the longer the time necessary for the effecting of such a thorough blending of blood and sputum as is observed when the expectoration is very thin (serous, for instance). The diffusion of the blood through the sputum is most complete in cases of fibrinous pneumonia. It is only in very confined spaces, such as are presented by the alveoli and finest air-tubes, that a uniform and intimate mingling of pathological secretions and blood can take place.

Blood disposed in *streaks* through the sputum does not usually proceed from the pulmonary parenchyma but from the upper parts of the air-passages, and gives no indication, therefore, of the nature of the morbid process going on.—*Specks* of blood frequently appear in the sputum, both in the earlier and later stages of caseous pneumonic infiltration.

The expectoration may also show by its red colour that it certainly contains blood, though at the same time no red blood-corpuscles can be detected under the microscope; in such circumstances it is probable that the corpuscles have undergone considerable modification, are possibly shrivelled up, and indistinguishable from the other figurate elements of the sputum, or that their disintegration has been complete and their colouring-matter in that way set free.

METAMORPHOSIS OF THE COLOURING MATTER OF THE SANGUINEOUS SPUTUM.

The longer the sanguinolent sputum is retained in the bronchi the more marked and extensive is the change in colour which it undergoes. It first becomes gradually reddish-brown, then yellowish-red, and finally loses all trace of red coloration; it subsequently takes on a yellowish hue, which deepens into saffron-yellow, yellowish-green, or even grass-green. All these tints are given to it by the *substances which are produced by the higher oxidation of the colouring-matter of the blood* (hæmoglobin); they represent the successive stages of that process,—a metamorphosis which is often observed in parts in which blood has been effused under the skin. Green is the last of the series of colours displayed by the hæmoglobin and its products when exposed to the continued influence of oxygen.—So long as the colouring-matter remains unchanged, and the expectoration continues distinctly and unmistakeably red, the blood-corpuscles are easily recognised, as they still possess their normal aspect and outline; but immediately the process of oxidation begins they are more or less altered in appearance, or may even be completely disintegrated, leaving behind them no visible trace of their previous existence in the sputum.—The first of the above-mentioned shades, reddish-brown or rust-colour, is characteristic of the sputa in the stage of hepatization in pneumonia, while the yellowish-red, or citron-yellow, or saffron-yellow tints mark the stage of resolution.

Greenish, or even *grass-green*, sputa (coloured thus, not by biliary pigment, but by altered hæmoglobin) occur sometimes in croupous pneumonia when it ends gradually (by lysis, not critically) in perfect resolution; in pneumonia also, which is followed by pulmonary abscess, and at the beginning of subacute caseous pneumonia (Traube). In all these affections the secretions are retained some time in the lungs,—usually for a period long enough to permit their colouring-matter to pass through all the grades of oxidation and become distinctly green; but in pneumonia of the ordinary type the sanguinolent sputum is coughed up and expelled before its hæmoglobin is so far changed as to become green, so that it is merely of a yellowish tint.

Microscopic examination of green sputa reveals the presence of yellow pigment, yellow pigmented molecules, and here and there yellow pigmented epithelial scales.

Sometimes in the warm summer months there is observed a peculiar variety of *yellow* sputum, the *egg-yolk* sputum (Traube, Löwer), in which the coloration seems to be due, not to the presence of modified hæmoglobin, but probably to the development of fungous growths. The first trace of it is usually noticed after the sputum has stood a short time in the vessel, and in the frothy layer at the surface; it may appear in any sputum of a frothy, tenacious character, and disappears when the weather becomes cooler or when the expectoration undergoes further change.

Under the microscope this sputum is seen to contain numerous masses of spores, resembling closely the leptothrix buccalis, and here and there also undoubted filaments of leptothrix. It is from the larger groups of spores that the yellow colour is derived. These fungi are probably introduced mechanically into the sputum as it passes through the mouth, and develope in the expectoration-dish under the favouring influence of heat. As the leptothrix is naturally of a somewhat yellow tinge, the colour is of course intensified by the multiplication of filaments. Microscopic examination has shown, also, that in the mouth and between the teeth of those patients whose sputum was of this nature were lodged large numbers of leptothrix thalli. Diagnostically and prognostically this "egg-yolk" sputum has not the slightest signification.—Another variety of sputum, identical with the foregoing in microscopic and other characters, differing from it only in colour,—which was grass-green, was observed by Rosenbach in a case of bronchial asthma; the coloration was developed only about 24 hours after the sputum had been expelled.

BILIARY PIGMENT IN THE SPUTUM.

The presence of biliary pigment communicates to the sputum various shades of colour, from yellow to green or even to deep grass-green. In appearance, therefore, they are absolutely indistinguishable from the above-described yellow or green sputa which, as already stated, derive their colour from the products of the higher oxidation of their hæmoglobin. A yellow or green coloration of the sputum is known to be due to biliary pigment only when the skin and mucous membranes have the same jaundiced hue, usually the result of a duodenal catarrh. The ordinary test for the presence of the colouring-matter of the bile, the occurrence of the well-known play of colours on the addition of nitric acid, is not satisfactory, as this same reagent produces a slight greenish tinge in the usually colourless mucous sputum.

This complication, the association of icterus (duodenalis) with a disease of the respiratory apparatus, is met with in the bilious form of pneumonia, though not in every case of bilious pneumonia is the sputum also necessarily yellow or green. On the other hand, it may happen that any other affection of the respiratory organs, simple bronchial catarrh, for instance, may be complicated by the occurrence of duodenal catarrh with icterus, when the sputum at once turns yellow or green; I have seen one well-marked case of this nature.

BLACK PIGMENTED SPUTA.

The sputum may be blackened throughout almost its entire extent (a somewhat rare event), or only in certain parts. This staining is commonly caused by *particles of carbon* which have been carried into the air-passages and mixed there with the fluid secretions; the greater the number of these particles the more intimate is their combination with the sputum and the deeper the shade of the black pigmentation.

Quantities of carbonaceous matter are often seen in the sputum of those who are exposed to the habitual inhalation of the sooty smoke of a badly-burning lamp; in such circumstances it is not unusual to find that the secretions coughed up in the morning are black. Small quantities of snuff also sometimes slip down from the nose into the air-tubes and are recognised in the catarrhal secretion as minute black particles. This black pigmented sputum probably occurs most frequently among those who work in coal-pits. In all these cases microscopic examination shows that the amorphous granules of carbon lie loose and free in the sputum; but they may also penetrate even into the substance of the lung itself, and are then incorporated with the epithelium of the air-vesicles and with the pulmonary texture.

Accurate investigations, carried on by Traube and Cohnheim, into the nature and surroundings of this carbon-dust have proved most clearly that it comes from without, and that it insinuates itself into the epithelium of the alveoli and into the interstitial tissue (in one case it had reached even to the bronchial glands); this is evident from the fact that the particles of which it is composed differ in no respect from those which float *freely* about in the atmosphere. In one of the two cases observed by Traube the specks of carbon found in the sputum resembled exactly those which the patient had habitually inhaled in the timber-yard in which he worked, their structural identity with the cellules of the pinus sylvestris being completely established. Böttcher has recorded an exactly similar case.

Absolute proof, however, that fine powdery matters, conveyed into the trachea, may be carried downwards into the air-cells and may work their way even into the tissue of the lung, is furnished by the experiments of Slavjansky. He introduced a certain quantity of cinnabar into the trachea of several animals, and afterwards found the particles of that substance in the epithelium of the alveoli, some of them scattered irregularly through the cells, others showing a definite and orderly arrangement; he discovered them also in the interalveolar septa, in the bronchial glands, and even in the blood, to which they had probably gained entrance through the lymphatic glands and vessels.

There is, further, sometimes seen in the sputum, a *black pulmonary pigment* (*Melanin*), in the form of *black, uniformly pigmented cells.* There is little doubt that this *Melanin*, which differs from all other organic pigments in being insoluble in alkaline lye and in resisting the bleaching action of chlorine, *is simply inhaled carbon-dust*, by which the pulmonary epithelium is stained of a uniform black colour. *Phthisis melanotica* is thus merely a chronic form of pneumonia, excited by the inhalation of carbonaceous particles.

Besides carbonaceous matters other variously-coloured particles, such as the dust of cinnabar or iron, are found in the sputum; these not only communicate their colour to the secretions but also give rise to more or less serious affections of the air-passages and pulmonary parenchyma (catarrh, or chronic pneumonic infiltration), which Zenker classifies as "diseases from the inhalation of dust." To the cases of siderosis pulmonum recorded by Zenker may be added one of great interest reported by Merkel, in which the lung was of a brick-red colour from a deposit of oxide of iron; the patient had been employed in a tile-work. Another patient, who had worked in an ultramarine factory and had suffered from chronic bronchial catarrh, brought up an exquisitely *blue-coloured sputum.*

The Sputa in Diseases of the Air-Passages.

Catarrh and inflammations of the respiratory passages, whether seated in the larynx or the larger or smaller bronchi, are always attended by the same species of sputum, as all parts of the mucous membrane have exactly the same structure.

At the beginning of an attack of catarrhal inflammation the sputum is simply mucous, consisting chiefly of a tough, vitreous, transparent mucus, with only a few figurate elements,—the mucus-corpuscles; in the expectoration-vessel it runs together and contains air-bubbles, and is therefore frothy and clear in colour. This is the *sputum crudum* of the ancients.—At a later stage of catarrhal affections it becomes richer in cells, contains, besides mucus and mucus-corpuscles, a few pus-corpuscles, and is thus of somewhat firmer consistence; as the inflammation abates the cell-elements are present in still greater numbers, the sputa take the form of irregularly globular masses (the *sputa cocta* of the older authors) and assume a dull yellowish colour. Both these stages, marked by a mucous and a muco-purulent sputum, are observed in every catarrhal or inflammatory bronchial affection; when the inflammation is of a croupous character, however, it is not *sputum,* in the ordinary acceptation of the term, that is expectorated, but chiefly the fibrinous bronchial casts already described on p. 180.—In putrid bronchitis and in bronchiectasis, also, the sputum has special characters, given in detail on p. 202.

The Sputa in Affections of the Lung-Substance.

The Sputum in Croupous Pneumonia.

The three stages of this disease are usually characterised by three distinct varieties of sputum.

1. In the *stage of engorgement* of the lungs the sputa, if present, are very scanty and tough, are composed mostly of mucus, include numerous bubbles of air, and are therefore transparent; they contain comparatively few morphological elements, are occasionally marked by streaks or spots of blood, and are of no definite

shape when expectorated. They coalesce in the receiving-glass, in which, on account of the large quantity of air enclosed in them, they float, forming a spumous layer on the surface of the water. The blood, which is present only in minute traces, is generally seen only on the surface of the sputum, and is not intimately mixed with it.—The expectoration of such a sputum, beset with specks of blood, is a point of great diagnostic importance, as by it pneumonia may be recognised some time before the physical signs of the disease are developed; lobular, central pneumonia is often first noticed, and distinguished from other acute affections of the respiratory organs by this symptom alone.

2. In the *stage of hepatization* increase of the plastic exudation in the alveoli and in the terminal bronchi is associated with a sputum more copious in quantity, exceedingly tenacious, less aerated, and more deeply tinged with blood; the latter is more intimately mixed with the mucus, and gives to it the *rust-colour* described as pathognomonic of the affection. The depth of the *coloration* varies with the relative amount of the blood and the length of time it lodges in the alveoli and bronchi. Reddish brown or rusty sputa are thus obviously of less recent date than those which are bright red. In the former are found, on microscopic examination, numerous altered blood-corpuscles, some distended, others shrivelled up and disintegrated, their colouring-matter being dissolved out and the round of oxidation-changes to which it is subject being already begun. The number of corpuscles seen under the microscope, therefore, is by no means proportionate to the intensity of the colour of the sputa when looked at with the naked eye. In bright red sputa, on the other hand, the histological structure and the colour of the corpuscles are preserved intact.—The *tenacity* of the sanguineous pneumonic sputum is such that the patient has often the greatest difficulty in getting it out of his mouth; it adheres to the sides of the crachoir and does not flow out when the latter is inverted. This toughness, and the transparency of the sputum, depend on the presence of mucus; increase of the number of the morphological constituents (blood-corpuscles and pus-cells) renders the sputum less transparent. The quantity of air, also, which is mixed in the sputum varies with the intimacy of the contact into which the respired air and the secretions are brought in the bronchi. Gradually, keeping pace with the advancing consolidation of the

lung and the consequent greater abundance of the pus-corpuscles, the sputum becomes less and less tough and adhesive, and is thus less confluent and more easily brought up; it begins rather to take on a more or less distinctly nummular or irregularly globular form.

Fibrinous clots, also, are found in the sputum, and constitute one of its most important elements; their presence indicates that the terminal bronchi are occupied by the same plastic exudation that fills the alveoli (see p. 180). They may be recognised while still in the expectoration-dish, on turning up the latter and examining the secretion which clings to its sides. On separating and washing these portions of the sputum with water the fine arborescent ramifications of the casts may be seen under a powerful magnifying glass.—Towards the end of the stage of hepatization the secretions become more fluid and less transparent, and the fibrinous coagula increase in numbers; the blood present in the sputum diminishes in quantity, its colour deepens into a dark reddish-brown, and in the event of there being no new exudation into any part of the lung all trace of bright-red, recent blood entirely disappears.

The various characters described so far do not always present themselves in the exact order mentioned, as it frequently occurs that after complete hepatization of one section of the lung another part, hitherto exempt from the inflammation, is attacked and goes through all the phases of the pneumonic process. Thus, whilst some of the sputa have the properties of those associated with the end of the stage of hepatization and the beginning of that of resolution, certain others, coming presumably from the portions of lung most recently affected, may be loaded with blood and be exceedingly viscid.

3. In the *stage of resolution* the *rust colour* fades, and the sputum becomes yellowish or of a darker, citron-yellow tint, from the metamorphosis of its hæmoglobin; it is expectorated with less difficulty, as it diminishes steadily in viscosity and no longer adheres to the sides of the vessel; it grows gradually more and more opaque, from augmentation of the number of pus-cells it contains, while the fibrinous bronchial casts disappear or break down in fatty degeneration. The secretions thus come to resemble closely those of simple bronchial catarrh. They are at first considerably more abundant than in the stage of hepatization, but

become by degrees scantier, more mucous, watery, transparent, and colourless; and finally, when the resolution of the pneumonia is complete, expectoration ceases entirely, or for a few days longer a scanty, mucous, bronchial secretion is discharged.

The features which have been enumerated as characteristic of the sputum in the various stages of pneumonia relate only to croupous pneumonia running an absolutely normal course. Should the disease, however, not end in resolution, but tend rather to fatal termination from exhaustion of the vital powers of the patient, or from pulmonary œdema, the ordinary pneumonic sputum is replaced by a secretion of more fluid consistence, containing numerous air-bubbles, extremely frothy, and often stained of a dark reddish-brown colour from the continued presence of blood (the *prune-juice sputum*).

In the rare cases in which the pneumonia results in *pulmonary abscess* a certain period usually elapses in which no sputum is expectorated; this is followed by a sudden and profuse discharge of greenish-yellow, perfectly purulent sputum, very fluid in consistence, and more or less offensive in odour, possessing all the properties of pus obtained from an abscess in any other situation.

This homogeneous sputum, when examined either with the naked eye or with the microscope, is found to contain numerous shreds of dead pulmonary tissue, consisting of elastic fibres, fat-crystals, crystals of hæmatoidin, and black pigmentary matter and micrococci.—Chronic pneumonia, also, occasionally terminates in the formation of abscess. In the purulent or muco-purulent sputa brought up at intervals from such an abscess, similar fragments of lung-tissue are seen, having a densely fibrous, cicatricial appearance, quite in keeping with the stony hardness of the diseased structure from which they come; elastic fibres, also, and plates of cholesterin (Leyden), are often present in this sputum.

When pneumonia runs into *gangrene of the lungs*, (not a common occurrence), the expectoration becomes excessively fœtid and of a dirty greyish colour, and is loaded with shreds of necrosed tissue and acicular crystals of the fatty acids (see p. 202).

The sputum of catarrhal pneumonia shows many points of difference from that of croupous pneumonia, the former affection being one which attacks primarily the bronchi, and spreads downwards so as eventually to involve the aveoli; of this nature is the broncho-pneumonia which occurs in children and the aged, and not unfrequently also in persons of middle age. As this form of pneumonia is not attended by any extravasation of blood the sputum presents the characters only of a catarrh, being simply muco-purulent, or containing at most, when the lungs are deeply congested, a few slight and transient traces of blood.

The Sputum in Tuberculosis and Phthisis of the Lungs.

Acute miliary tuberculosis of the lungs gives a sputum in no respect different from that of simple bronchial catarrh.

The sputum in *chronic* cheesy degeneration, before the process of excavation has begun, consists almost exclusively of the catarrhal secretion of the bronchial mucous membrane, and is consequently indistinguishable, with the naked eye at least, from that of ordinary catarrh. On microscopic examination, however, elastic fibres are sometimes seen, an observation which of itself is conclusive evidence that a destructive process is going on, even in the absence of the usual physical signs of such a condition. The presence of pulmonary epithelium in the sputum, on the other hand, at one time looked upon as an infallible indication of that form of phthisis which is essentially a desquamative pneumonia, is not entitled to be so regarded, as pulmonary epithelial scales similarly altered in structure are occasionally found in the sputum of simple catarrh.

The quantity of the sputum generally depends on the intensity and extent of the bronchial catarrh accompanying the phthisis; it is therefore scanty at the outset of the disease, and becomes more abundant in its later stages. The expectoration may also be completely suppressed, at least for a time, particularly in case of remission or definite arrest of the progress of the disease.—Occasionally there is blood mingled with the secretion, in reddish specks or streaks, seldom in the form of a generally diffused coloration; if the admixture be of long duration or of frequent recurrence we may infer, with almost absolute certainty, the existence of chronic caseous infiltration of the lungs, even when the physical signs which mark this affection cannot be distinctly elicited.

On the formation of cavities in the lung-substance the sputa at once take on more specific characters; they are of firmer consistence, assume a rounded or nummular form, become more or less ragged at the edges, opaque, of a yellowish-green or dirty grey colour, contain little or no air, and sink in water (the *sputa rotunda, fundum petentia* of the early physicians).

Now and then the cavernous sputum contains blood, recently effused or of older date, disposed either in a thin layer on its surface or intimately mixed up in its substance; the secretion then presents a more or less intense coloration, varying from light red to reddish brown. The blood in this case has evidently leaked from the smaller vessels, whose walls are disorganised and broken down by the extension of the morbid process. In this sputum is usually also included a variable quantity of catarrhal bronchial and buccal secretion, which, being very thin and full of air-bubbles, floats on the surface of the fluid in the expectoration-glass. The larger the quantity of this catarrhal exudation and the more crowded it is with air-bubbles, particularly if it be brought up with considerable pain and effort, the greater difficulty is there in distinguishing between it and the proper cavernous sputum. Very frequently the latter, in these circumstances, does not sink to the bottom of the vessel, but is suspended in the general mass of expectorated matters, in the frothy, mucous layer near the top.

On microscopic examination of the cavernous sputum there are found multitudes of pus-cells (hence its opacity), free nuclei, detritus, and occasionally elastic fibres.

The cavernous sputum, fluctuating in quantity from time to time according as the morbid process is aggravated or arrested, retains for a considerable period the physical properties above described; but if the excavation be occupied by a profuse fluid secretion which stagnates there for some time, the sputa become of a dull muddy colour, acquire a somewhat offensive odour, diminish in consistence, lose all appearance of having any definite shape, and tend to coalesce in the crachoir.

In the species of consolidation of the lungs which do not lead to phthisis,—for example, in atelectasis of the lungs, due most commonly to prolonged compression by pleuritic exudation, or more rarely (when it is usually only partial) to compression arising from some other cause, and, further, in the forms of collapse dependent on the blocking-up of large bronchi, and in condensation from hypostasis,—no characteristic sputum is discharged, but only such as owes its existence to the catarrh which accompanies these affections.

The Sputum in putrid Bronchitis and in Gangrene of the Lungs.

These two diseases have at least one feature in common,—there is associated with them a certain destruction of pulmonary tissue, the result of a putrefactive process, which, again, is probably set up in consequence of the introduction of bacteria or spores into the air-passages; as might be expected, therefore, the sputa also present some characters which are common to both. These, enumerated by Traube, are as follows: the sputa are very abundant, have a somewhat fluid consistence and a dirty greenish yellow colour, and separate into three strata on standing; the uppermost layer is greenish yellow, opaque, and frothy; the middle layer is strikingly transparent, albuminous, and almost serous in consistence; the undermost layer is yellow and opaque, and is composed almost exclusively of swollen pus-corpuscles and a detritus which contains a number of dull yellowish-white, soft *cores*,* whose size varies from that of a grain of millet or oats to the bulk of a bean; these cores have also an excessively *fœtid* odour, and contain the *needle-shaped crystals of the fatty acids* described on p. 182.

The chief condition which seems to be necessary to the formation of these crystals is that the sputum should be retained for some time in the ulcerated gangrenous bronchi or cavities; this occurs particularly in sinuous excavations not in communication with any of the larger bronchi. Fatty crystals are therefore wanting in the secretions of a large cavity opening freely into a bronchus of considerable calibre, as in such a case the sputum is always at once ejected. In the same way also may be explained the fact that putrefaction takes place so readily in bronchiectatic cavities while it is so exceedingly rare in those of phthisical origin; bronchiectatic cavities are situated in the *lower* lobes of the lung, and their secretions are brought up and expelled with greater difficulty, and therefore remain stagnant for a much longer period than those of phthisical cavities, which are generally situated in the *upper* lobes.

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* Traube recognises four different stages in the development of these cores. In the beginning of the affection, when they are first discovered, they for the most part consist of pus-corpuscles and detritus; at a later period they become dirty grey, a colour which they always afterwards retain, while the débris enclosed in the purulent mass shows globules of fat. In the third stage they are composed chiefly of débris, within which not only the fatty globules but acicular crystals of fat are observed. In the fourth stage they are greatly increased in number and gathered together into bundles.

The sputum of pulmonary gangrene and that of the putrid stage of bronchiectasis are distinguished from those of other affections involving destruction of tissue (caseous pneumonia, pulmonary abscess) by containing *few or no elastic fibres.* The latter seem to be completely destroyed by the action of the putrefactive material, the chemical properties of which, however, are still unknown; the connective tissue of the lung, on the other hand, is not in any way affected by this material.

The element which effects the decomposition of the elastic fibres is evidently present in the sputum, as the filtrate of gangrenous sputa, while alkaline in reaction, completely dissolves elastic tissue (taken, for example, from the ligamentum nuchæ of the calf) and boiled white of egg in 1—4 days, but has no influence whatever on gelatin-yielding tissues; this process is therefore exactly analogous to that which goes on within the lungs. The material on the presence of which this action depends appears to be of the nature of a ferment. It, too, is subject to disorganization, as the filtered gangrenous sputa no longer have the power of dissolving elastic fibres when, at the end of a few days, they become cloudy and undergo further decomposition. The contents of gangrenous cavities also, after removal from the dead body, and indeed all other putrid fluids whatsoever, proved equally powerless to effect the solution of elastic tissue (Filehne).

Chemical examination (by Jaffe) has shown that ammonia, sulphuretted hydrogen, leucin and tyrosin, volatile fatty acids (butyric acid), enter into the composition of the sputum in pulmonary gangrene and putrid bronchitis.—On being introduced into the trachea of animals, the gangrenous cores described above convey infection to the healthy tissues, if not at once expelled by coughing. This is usually followed by the occurrence of local, circumscribed pneumonic inflammations (particularly in the case of rabbits), or even by gangrene of the lungs (Leyden and Jaffe). The inflammation seems to be really due to the presence of vegetable parasites, which have been discovered by Fürbringer in great numbers in the gangrenous masses; the fungi consisted chiefly of the aspergillus niger and mucor.—These same fungous growths and, under certain circumstances, even acicular fatty crystals, like those of the sputum in pulmonary gangrene, may be developed in a simple catarrhal sputum which has been exposed some time to the air.

The Sputum in Bronchiectasis.

It has the characters of the muco-purulent sputum, is generally yellowish-green or dirty greenish-white in colour, homogeneous and confluent. In colour and consistence, as in its microscopic elements, it thus differs in no respect from the expectoration in chronic bronchial catarrh; in both cases it is the result of hypersecretion from the bronchial mucous membrane. Since, however, the bronchial secretion stagnates for a longer or shorter period in the dilated bronchi, not only when the latter are cylindrical in shape but also when they are sacculated, both of which conditions are generally present together, it acquires an *offensive odour*, which frequently has a certain resemblance to that which pervades a soap manufactory; the smell is most penetrating when the sputum is recently expectorated, and becomes less intense when the secretion has stood some time in the vessel.—The bronchiectatic sputum is, further, brought up only at long intervals, and then in large quantity. As the sensibility of the walls of the bronchiectatic cavity is greatly diminished the fluid accumulates till it reaches the orifice of the communicating bronchus; violent coughing is now excited and the sputum is ejected, when the patient has rest again for a period usually of several hours, expectorating at most only a little catarrhal sputum, till the cavity is filled anew, and the same process is repeated.

The quantity of sputum discharged in the twenty-four hours may amount to several hundreds of grammes; it is usually somewhat greater in the morning, as the secretion gathers in the cavity during the night. The fœtid odour and the periodical evacuation of a large quantity of sputum, serve to distinguish the bronchiectatic secretion from that proceeding from phthisical excavations.

In the expectoration-dish the bronchiectatic sputum usually separates into two or three layers, the upper of which is transparent and very fluid, the lower opaque, almost exclusively purulent, and resting on the bottom of the glass; the middle stratum consists chiefly of a quantity of flocculent mucus.

On the subsidence of the bronchial catarrh which attends every case of bronchiectasis, considerably less fluid is secreted, and as expectoration becomes more easy the sputum loses its fœtor, and is then almost identical in character with the muco-purulent sputum of bronchial catarrh. If, on the other hand, simple bronchiectasis is followed by ulceration of the dilated bronchi, and if, further, under the influence of minute vegetable or animal organisms, which find their way into the air-passages and there multiply and develope, a putrefactive decomposition of the secretions sets in (particularly in summer), the needle-shaped crystals of the fatty acids, mentioned on p. 202 when discussing putrid bronchitis and pulmonary gangrene, are found in the sputum.

EXAMINATION OF THE ORGANS OF CIRCULATION.

Inspection of the Præcordial Region.

The contraction of a heart which is structurally sound, normal in position and acting quietly, usually manifests itself externally as a slight elevation of the tissues in the fifth intercostal space, between the parasternal and mammillary lines; it is synchronous with the systole, does not raise the skin above the general level of the ribs, and is perceptible only over a small, circumscribed area, 1½—2, or at most 2½ ctm. in breadth. The normal impulse, commonly called the *apex-beat*[*] of the heart, never passes, to the right or left, beyond these limits. In children, however, it is not always situated in the fifth, but sometimes rises as high as the fourth, intercostal space, the diaphragm being drawn upwards with greater force by the lungs; in children also it not unfrequently passes a short distance (almost 1 ctm.) over the mammillary line towards the left side. On the other hand it occasionally happens, though relatively seldom, and only in the aged, that the heart's impulse is seen in the sixth intercostal space; in this case the displacement is due to diminution in the attractive force of the lungs and in the elasticity of the large vessels springing from the heart.

The situation of the apex-beat varies with the rise and fall of the diaphragm in the movements of respiration and on turning over towards the left side. The influence of respiration in altering the position of the heart's impulse is noticeable only on making a very deep inspiration, when the apex sinks, sometimes even behind the sixth rib, so that, the opposition to the transmission of the stroke of the heart on the chest-wall being increased, the heaving impulse can no longer be felt; in expiration it mounts again to its normal level. In quiet respiration no change takes place in the situation of the cardiac impulse. Lying on the left side brings it a little over the left mammillary line, occasionally as much as 2 ctm.; on turning on the right side no displacement, or the very slightest, is observable.

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[*] By the term *apex-beat* is understood not only the impulse of the actual apex, but also that of the lower segment of the heart. The proper *apex* does not lie in the fifth intercostal space, but behind the sixth rib, and is also covered in front by a tongue-shaped process of pulmonary tissue connected with the lower border of the left lung.

The apex-beat is not always visible, but is generally perceptible to the finger pressed deeply into the intercostal space; it escapes the eye also when the heart is acting very feebly, when the chest-wall is rigid and covered by a thick layer of fat and powerfully developed muscles, when the intercostal spaces are narrow, and when the heart, during full inspiration, is overlapped by the margin of the lung. (For the pathological conditions which cause the disappearance of the apex-beat, see p. 213).

In addition to the apex-beat, and occasionally also when it is wanting, a *diffuse* impulse, or undulation of the tissues in the præcordial region, may be observed, more particularly when the heart's action is abnormally strong.—A certain amount of vibration is also often felt between the third and sixth costal cartilages and over the lower portion of the sternum; it arises from the systolic tension of the mitral and tricuspid valves, and is designated the *valvular impulse*.

Vivisection teaches that the finger placed on *any* part of the exposed heart's surface experiences a distinct shock on the occurrence of each contraction; but in normal circumstances, from the relative position of the heart and lungs, only the stroke of the *apex* is felt. The entire base of the heart is covered by lung, which renders the transmission of its impulse to any distance a matter of difficulty; and this difficulty is increased by the backward movement of the base at each systole, by the resistance offered by the ribs, and by the thickness of the thoracic parietes (including the pectoral muscles and adipous tissue) in that region. The apex, on the contrary, is in immediate contact with the chest-wall, is formed chiefly by the powerful muscles of the left ventricle, and lies behind the yielding soft parts of the intercostal space; the most important consideration, however, is that this portion of the heart is tilted forwards at each systole, so that the tissues over it are of necessity raised along with it.

If the forementioned conditions, described as unfavourable to the occurrence of a visible impulse over the base of the heart, be removed, the apex-beat is accompanied by a distinct base-beat; this is the case in children with thin and yielding chest-walls, in all cases in which the heart, in consequence of retraction of the anterior margin of the left lung (from atrophy), is in close contact with a large part of the thoracic parietes, and in all hypertrophies of the heart, especially of the left ventricle.

In a few instances a *double impulse*, accompanying with more or less regularity each systole, has been observed (Skoda, Bamberger, Leyden). It occurs in aggravated cases of mitral insufficiency, and arises from the non-coincidence of the contractions of the two ventricles. To the *first* of these strokes alone the pulsation in the arteries corresponds, with the second it is wanting. This non-simultaneous contraction may be explained in the following way: when the mitral valve is markedly incompetent the overfilled right ventricle is unable to empty itself completely during the systole, and the next instant (during the diastole) is again distended with blood and so excited to renewed contraction; the left ventricle, on the other hand, takes no part in the second phase of the contraction of the right heart, as it contains at this stage but a small quantity of blood, or if it does act in concert with the second contraction of the right ventricle, it does so with greatly diminished force.—It is quite conceivable that the so-called abortive contractions, that is, those which are so feeble as to produce no arterial pulse, often associated with grave mitral lesion, arise from the non-simultaneous contraction of the two ventricles; though if this assumption were correct a double impulse should accompany the cardiac systole, which is not actually the case.

Cause of the Heart's Impulse.

During the diastole the intracardiac pressure is equal at all points on the inner surface of the ventricular walls, but when the heart contracts and the blood is thrown into the great vessels this pressure suddenly becomes less over the heart's outlets than at the part diametrically opposite to them,—the apex of the heart; the latter, therefore, in consequence of the *recoil* so generated, moves downwards and forwards at each systole. It is the operation of the same force, the sudden development of a difference in pressure, that sets in motion Segner's water-wheel, that produces the recoil which follows the discharge of firearms, and that causes a freely-suspended cylinder, filled with water and provided with an escape-pipe at its lower end, to move backwards, in a direction opposed to that of the jet of water, when the stopcock is opened (Gutbrod, Skoda). But the recoil-theory explains only the impulse of the apex of the heart, and not that of the base; the latter arises from the *hardening* *and swelling of the heart at the beginning of the systole* (Arnold, Kiwisch, Ludwig).

It appears to me, therefore, that the theory of Gutbrod and Skoda and that advocated by Arnold, should not be regarded as antagonistic: each, taken *alone*, is capable of explaining only certain of the pathological phenomena dependent on the heart's action; taken *together*, they satisfactorily explain them *all*.

The essential appearances seen on exposing the pulsating heart by vivisection are: *locomotion downwards and forwards* combined with rotation from the left side towards the right, and *increase in thickness* during each systole; *all* these factors must be taken into account in studying the causes of the cardiac impulse, no theory being free from objection which is founded on only one of them. While it is generally admitted that the impulse of the base of the heart is due simply to systolic increase in thickness and firmness of the cardiac tissue at that part, authorities are far from being unanimous in their explanations as to the manner in which the downward and forward movement of the *apex*, which gives rise to the apex-beat, is brought about. That the apex of the heart actually does move in the direction described, and at the same time rotates from left to right, has been frequently demonstrated, both in animals and in men suffering from thoracic fistula in the præcordial region. Bamberger teaches that the change in the position of the heart is the result of the systolic stretching of the aorta and pulmonary artery, and Kornitzer that the movement of rotation depends on the somewhat spiral arrangement of these vessels as they spring from the heart,—the elongation which they undergo making this spiral to turn slightly round on its vertical axis, carrying the heart with it. But Kornitzer's further hypothesis that this rotation has the effect of tilting the apex of the heart forwards, and should therefore be ranked as the special cause of the cardiac impulse, has not yet been satisfactorily proved. The same objection applies to another theory, more recently introduced by Aufrecht, which finds the cause of the heart's impulse in the *systolic flattening of the aortic arch*, and which seeks to explain exclusively on this ground all the physiological and pathological phenomena connected with the heart's beat; the observation that with each systolic discharge of blood into the aorta a diminution of the curvature of that vessel takes place, the aorta itself rising and the base of the heart sinking, is essentially corroborative of Bamberger's theory that the locomotion of the heart downwards is produced by the systolic stretching of the vessels, Aufrecht, however, holding that the pulmonary artery takes no share in the production of these changes. But whilst Bamberger does not attempt to found any doctrine as to the cause of the heart's impulse on this systolic depression of the heart by the elongation of the great vessels, Aufrecht argues that to it is added a forward movement of the organ, and that these together give rise to the impulse. The two theories thus agree so far, in regarding the injection of the blood into the arteries as the cause of the apex-beat (the latter, indeed, instantly disappears, as Hiffelsheim and Jahn have shown, on cutting off the supply of blood to the chambers of the heart), but differ concerning the manner in which this cause operates.—The view which has found most wide acceptance is that advanced by Gutbrod and adopted by Skoda, based on the physical principle of recoil.

The two most important objections to the Gutbrod-Skoda theory are the following: 1st, Bamberger, Kürschner, Scheiber, and others, maintain that the physical principle of recoil is not applicable to the heart, as those parts on which the force of the recoil should fall are precisely those which by their contraction generate the original force; a recoil, therefore, at the apex would be overcome by the counterpressure arising from the contraction of the heart from the apex towards the base.

This counterpressure, however, is in part neutralized by the downward movement of the heart at each systole, but chiefly by the fact that the heart, as Skoda points out, does not contract simply from the apex towards the base, in the direction opposed to that of the recoil, but *concentrically*, so that the increase of pressure on its walls is everywhere *equal;* in this case the cardiac parietes are placed under exactly the same conditions as the sides of a cylinder filled with water, —they are subjected to a uniform pressure.

2ndly. The second objection rests on the observation that that point on the inner surface of the heart which is diametrically opposite to the arterial orifices does not coincide with the apex, but with a spot on the wall of the right ventricle (Scheiber); the recoil from the orifice of the pulmonary artery takes effect on the side of the right ventricle, that from the aorta exactly on the apex of the heart, so that the resultant of these two forces passes through a point situated somewhat to the right of the apex. It is only in cases of unequal hypertrophy of the different parts of the heart that these relations are appreciably disturbed.

Skoda meets this objection by the statements that the aortic and pulmonary orifices do not lie in the same plane; that the heart may, on account of the intricate disposition of its muscular fibres, be considered as a single-chambered tubular organ, the lower part of which, diametrically opposite to the arterial orifices, suffers a certain amount of pressure from the current of blood flowing from it into the great vessels, the area so affected being equal to the sum of the transverse sections of the pulmonary and aortic openings; and that this pressure is distributed over the surface of the lower segment of the heart, which assumes a conical form during the systole; and he concludes that it is not essential to the general accuracy of his theory that the force of the recoil should light *exactly* on the *apex.* Jahn has also recently shown that the resultant of the lines of recoil from the aorta and pulmonary artery falls precisely on the apex of the heart, as indicated by Gutbrod and Skoda.

Hiffelsheim has demonstrated the recoil experimentally in a caoutchouc heart filled with water, discharging into an artificial aorta; he found that the force of the recoil is proportional to the volume of fluid contained, to the thickness of the walls of the artificial heart, and to the diameter of the aortic orifice.

On p. 214 have been mentioned certain clinical facts which admit of satisfactory explanation by means of the Gutbrod-Skoda theory only.

PATHOLOGICAL ALTERATIONS OF THE NORMAL CARDIAC IMPULSE.

These affect the situation, force, and extent of the impulse.

Alteration in the *situation* of the apex-beat arises from *displacement* of the whole heart, which is very frequently the result of change in the position of the diaphragm,—when the latter is depressed the heart sinks, when elevated the heart rises.

Depression of the whole diaphragm may be caused by bilateral emphysema of the lungs, lowering of one-half by the presence of gas or fluid in one of the pleuræ. The dislocation of the heart in pulmonary emphysema takes place downwards and to the right, in effusion into the left pleura, when the fluid is moderately abundant, simply downwards, or when the quantity is excessive, to the right side, sometimes even as far as the right mammillary line; pyo-pneumothorax of the left side has the same effects, though not usually to such a marked degree. On the absorption of the exudation, should the left lung fully expand as the fluid is removed, the heart, if it have not formed adhesions to its new surroundings, returns gradually to its normal position.

If the displacement of the heart towards the right be but slight the apex-beat remains at the natural level or, if the diaphragm be forced a little downwards by the weight of the fluid, sinks to a trifling extent; the position of the heart with regard to the direction of its axis usually continues normal, however, that is, the apex is still the part which lies furthest to the left. But as the heart approaches the sternum its axis becomes more and more vertical; if it be thrust beyond the middle line of the sternum the apex rises, from the greater elevation of the *middle* segment of the diaphragm,—the part over which the apex must, of course, pass when pushed over into the right side of the thorax; the impulse may then be felt in the fourth right intercostal space. The apex forms now the portion of the heart situated furthest to the right, as its movement is not hampered by its anatomical relations, whilst the base, from the manner in which it is bound down by the great vessels, is less capable of being altered in position.—Very copious effusions into the *right pleura* push the heart beyond its normal limits towards the left side, sometimes even as far as the axillary line. In most cases also in which the heart is displaced by pleuritic exudation the apex-beat is considerably augmented in force, on account of the greater resistance offered by the fluid to the emptying of the right ventricle; the pulsation not only of the apex but commonly also that of a large portion of the heart's surface may then be seen.

The diaphragm may come to occupy an abnormally high position as the result of atrophy of one lung, or of atelectasis of a lung after absorption of pleuritic exudation of long standing; or it may be driven from below upwards by increase in the volume of the abdominal organs, particularly by hepatic, splenic, uterine, and ovarian tumours, and by ascites or meteorism. The heart rises higher in the thorax in proportion to the extent of the upward displacement of the diaphragm, and in extreme cases may pulsate in the third intercostal space.

Contraction of the left lung causes the heart to beat not only at a higher level but also frequently to the outside of the mammillary line, or even in the axillary line, as the mediastinum, like the diaphragm, is also dragged further into the diminished cavity of the thorax. In contraction of the right lung the heart, with the anterior mediastinum, passes over to the right side, sometimes as far as the right border of the sternum and even beyond it.

Congenital malposition of the heart exists in cases of *Inversion of the Viscera;* the heart is here found on the right side, beating in the fifth right intercostal space, and the cardiac axis is directed from above and behind downwards, forwards, and to the right.

The position of the apex-beat (or cardiac impulse) may further be modified by *increase of the size of the heart,* hypertrophy with dilatation.

When the *left* ventricle is hypertrophied and dilated it is chiefly in its *long* diameter that its size is augmented, so that the impulse is shifted downwards to the sixth, seventh, or even the eighth intercostal space; as the ventricle also gains sensibly in breadth the pulsation of the heart extends outwards beyond the mammillary line.

In hypertrophy with dilatation of the right ventricle it is principally the *breadth* of the heart that is added to; its impulse, accordingly, passes further to the right than normally, sometimes to the right margin of the sternum or even to the right mammillary line. In cases in which the dilatation of the right ventricle is moderate in amount the cardiac impulse to the right is not so distinctly visible as it is to the left when the left ventricle is hypertrophied to an equal degree, the intercostal spaces becoming considerably narrower towards the sternum.

In dilatation of the right heart the cardiac impulse oversteps the normal boundaries also to the left and downwards; it reaches outwards to or even beyond the left mammillary line, and inferiorly often to the sixth intercostal space. Both these conditions are due chiefly to a change in the relation of the axis of the heart to that of the body: the heart, when the dilatation of its right half is considerable, assumes a less upright and more *horizontal* position.—In very pronounced cases of hypertrophy and dilatation of the left side of the heart the impulse, though most marked towards the left, is appreciable beyond its normal limits also towards the right.

The *force of the heart's impulse* presents great differences even amongst individuals who are in perfect health, and is found also, other circumstances being the same, to vary chiefly with the energy with which the heart contracts. The impulse may be so *weakened* as to escape detection either by eye or hand, or may, on the contrary, become so *forcible* as to elevate and throw into vibration a large part of the chest-wall.

Enfeeblement of the cardiac impulse, even to such a degree that no perceptible shock is communicated to the hand, takes place (apart from the physiological causes already mentioned on p. 207) in the following circumstances:

1. When the heart's action becomes less vigorous than in health. This may occur as the result of fatty degeneration of the cardiac muscular fibres, or of inflammatory changes in the latter, due to myocarditis or arising in the course of severe acute affections; or it may be owing to the prolonged influence of a high febrile temperature (from the so-called albuminous infiltration of the muscular fibres), or to abnormally feeble innervation of the heart. This disorder of the innervation may be of a transient character, as in an ordinary case of fainting, or it may be more lasting or recurrent, presenting itself frequently as a concomitant symptom in many diseases of the nervous system; and, finally, it usually appears shortly before death, indicating paralysis of the nervous system.

2. When the heart is separated from the chest-wall by any medium, which may either be interposed between that organ and the pericardium (—the presence of fluid, or rarely air, in the

pericardium), or which covers the heart (an emphysematous lung, for instance), or which comes between the heart and the thoracic parietes (such as pleuritic exudation, air in pneumothorax). Displacement of the heart from the causes just mentioned does not abolish the cardiac impulse, the latter being in such conditions usually visible at some other than the normal spot.

3. When the heart has contracted adhesions to the pericardium. In these cases, as the systolic movement of the heart downwards and forwards is rendered impossible, the impulse is absolutely wanting, and instead of the raising of the tissues by the apex-beat we have systolic retraction taking place in the region where the former is normally felt, (see p. 222).

Finally, it is sometimes observed that when stenosis of the aortic orifice or of the left auriculo-ventricular orifice reaches a certain degree of intensity *the apex-beat is absent ;* in such circumstances also *the impulse of the base* is, on account of the consecutive hypertrophy, much increased in force and spread over a larger surface than naturally.

The absence of the apex-beat in severe stenosis of the aortic orifice admits of very satisfactory explanation by means of the Gutbrod-Skoda theory of the causation of the normal impulse, and in the following way :—the force of the recoil depends, not only on the energy with which the ventricle discharges its contents into the arterial system, but also on the rapidity and volume of the current of blood, that is, on the diameter of the aperture of exit. When this diameter is encroached upon in stenosis the quantity of blood propelled from the ventricle in a given unit of time becomes less, so that the diminution in pressure at the aortic orifice is not so marked, nor is the systolic *difference* between the pressure at the aortic orifice and that at the apex of the heart so great, as under normal conditions ; the recoil of the apex, therefore, that is, its systolic locomotion downwards and forwards, is also less, and may become so slight as to be no longer appreciable.

In like manner the want of the apex-beat in severe stenosis of the left auriculo-ventricular orifice may readily be accounted for ; the constriction prevents the left ventricle being so completely filled with blood as it should be before its contraction, and the systolic difference between the pressure over the aortic orifice and that at the apex of the heart is consequently *abnormally small.*

The force of the cardiac impulse is increased by whatever strengthens the heart's contractions : in health, therefore, this takes place from mental excitement or violent physical exertion ; pathologically it occurs in all febrile conditions, in inflammatory diseases of the heart, endocarditis, pericarditis (in the latter, however, only so long as the exudation is insufficient in quantity to obscure the perception of the shock), in the various neuroses of the heart (which may exist independently of any other disease, or may complicate other morbid processes of the most diverse kinds), and in all those conditions which favour the transmission of the heart's impulse to the surface, such as condensation of the upper lobe of the lung, retraction of the margin of the left lung, &c. This phenomenon is met with most often and in its most aggravated form, as the result of increase of the muscular substance (*hypertrophy*) of the heart.

The impulse is the more forcible the more the hypertrophy of the muscular structure of the heart preponderates over the dilatation of its cavities; should the hypertrophy cease to increase, or should it even decrease, as it frequently does in the more advanced stages of the disease, from fatty degeneration of the muscular fibres, so that the dilatation comes to be a more prominent feature in the case than the hypertrophy, the impulse of the heart grows feebler; this is observed in the later stages of all cardiac lesions.

The heart's impulse is stronger in hypertrophy of the *left* ventricle than in that of the right. When the power of the *left* side of the heart is increased to a considerable degree the impulse takes on a *heaving* character, and in severe cases the greater part of the front of the left side of the chest is elevated and thrown into distinct vibration each time the heart contracts; in the diastole the raised portion returns sharply and with some force to its original position. Such a heaving impulse is never observed in hypertrophy of the right ventricle, as in the latter the thickening of the cardiac muscle is not so great as in the left ventricle; while the thickness of the hypertrophied wall of the right heart amounts only to ¾—1 ctm. or very little more, that of the hypertrophied left heart reaches 2—2½ ctm., or even slightly exceeds that measurement. In a large number of cases also an increase in the force of the cardiac impulse from hypertrophy of the right ventricle is not very readily detected by the eye, as the right side of the heart is not so favourably situated anatomically as the left for the conduction of its stroke to the surface (compare p. 212); it may, nevertheless, always be *felt*, by placing the hand on the lower part of the sternum.—Other things being equal the impulse of the heart is most powerful in enlargement of the whole organ.—When the hypertrophy of either ventricle is considerable it may give rise to very marked *prominence* in the præcordial region, particularly in young persons, whose thoracic parietes yield readily to pressure.

The following is a short statement of the general causes of hypertrophy of the heart.

Hypertrophy of the heart is almost invariably the result of the existence of obstacles to the free circulation of the blood; to overcome these obstacles more energetic contraction of the heart is necessary, and this, as in every case in which a muscle is habitually called upon to exert its full power, leads to an increase in the bulk of the cardiac muscular substance. The development of hypertrophy is always preceded by *dilatation* of that ventricle which, on account of the resistance encountered in the systemic or pulmonary circulation, is unable to empty itself completely of its blood at each contraction. Hypertrophy is thus always associated with dilatation of the corresponding ventricular cavity. This is known as *excentric* hypertrophy.

Hypertrophy (with dilatation) of the *left* ventricle arises from the presence of some *impediment to circulation in the aortic system*, hypertrophy (with dilatation) of the *right* ventricle from *obstacles in the pulmonary system*. Other conditions being similar, the thickening is usually the more marked the nearer the obstruction lies to the ventricle.

Hypertrophy of the *left* ventricle may be caused by atrophy of the kidneys, by sclerosis of parts of the aortic system, by *atheromatous degeneration* of the walls of the aorta, by *stenoses* of the aorta, by *aneurisms* of the aorta (when complicated by lesions of the aortic valves), by *insufficiency* of the aortic valves, and *stenosis* of the aortic orifice.

Hypertrophy of the *right* ventricle may be due

1. To *engorgement of the pulmonary circulation*. It is in this way, by impeding the return of the blood through the pulmonary vein, that *mitral lesions* (insufficiency and stenosis), cause enlargement; in order to enable the right ventricle to rid itself of the whole of its contents, notwithstanding the opposition presented by the overloaded condition of the pulmonary vessels, its muscular mass undergoes large increase.

2. To *obliteration of a large number of the pulmonary capillaries*. This arises from vesicular emphysema, atrophy of the lung from chronic interstitial disease, prolonged compression of the lung by pleuritic exudation, and from lateral and angular curvatures of the spinal column. In these cases it is inevitable that the right ventricle should undergo a certain amount of hypertrophy, that it may contract

with sufficient force to open up a pathway for the blood through the already reduced area in which it circulates in the lungs.*

3. To *valvular lesions at the orifice of the pulmonary artery* (stenosis of the conus arteriosus or of the ramifications of the pulmonary artery, insufficiency of the *semilunar valves* at the root of the same vessel,—both very rare affections).—Incompetence of the tricuspid valve is usually associated with hypertrophy of the right ventricle, which, again, is chiefly the result of the *concomitant mitral lesion*, and which, in uncomplicated cases, is exceedingly slight.

If the circulation be obstructed both in the aortic and the pulmonary systems, (as in cases of lesion of the aortic and mitral valves), hypertrophy of both ventricles takes place. Severe aortic lesions alone may occasion thickening of the right as well as of the left ventricle, as in the more advanced stages of aortic valvular disease congestion of the pulmonary circulation also appears.

The *auricles* also *undergo hypertrophy* when, from contraction of the auriculo-ventricular orifice, they are unable to empty themselves entirely, or when, from insufficiency of the auriculo-ventricular valves, the blood regurgitates at every systole from the ventricles into the auricles, so that the latter are kept constantly in a state of overdistension; in both affections the complete evacuation of the auricles becomes impossible, and conditions are presented which are favourable to the development of hypertrophy of these parts, (of the left auricle, for instance, in stenosis and insufficiency of the mitral valve).

Hypertrophy with dilatation of the ventricles may also arise *independently* of the existence of any mechanical hindrance to the circulation, and due simply to overaction of the heart. Though the truth of this doctrine has often in the past been held doubtful it has recently been firmly established on the strength of numerous and accurate observations. This form of hypertrophy without any trace of valvular lesion is developed especially after violent and prolonged physical exertion, as in soldiers as the result of long and fatiguing marches, &c., and amongst the labouring classes; its production is also sometimes favoured by undue indulgence in spirituous liquors and by immoderate smoking.

EXTENT OF THE HEART'S IMPULSE.

An impulse whose breadth within the fifth intercostal space amounts to more than 2½ ctm., and which thus passes beyond the mammillary line to the left or the parasternal line to the right,—provided there be no displacement of the organ as a whole,—invariably indicates hypertrophy of the heart.

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* Bäumler has recently made the assertion that extensive, but more particularly complete *adhesion of the pleural surfaces* to each other gives rise to a disposition to the development of *hypertrophy of the heart*, as the free expansion of the various parts of the lungs is thus hampered or prevented and the elasticity of the lungs diminished. But as diminution of the elasticity of the pulmonary tissue impedes the outflow of blood from the pulmonary veins towards the left ventricle the pulmonary circulation becomes overloaded, and the work which the right heart has to perform is increased. There is thus produced a *disposition* to hypertrophy of the right heart, the occurrence of which in such cases is generally further favoured by other concomitant circumstances.—When at a later stage the congestion extends also to the systemic veins, and the left ventricle is thus called upon to contract more energetically, the left side of the heart also becomes enlarged, though not usually to such a considerable degree as the right.

The heart's impulse in hypertrophy may be distinctly appreciable, both to the eye and hand, in two or even three intercostal spaces at once; when the enlargement is considerable and limited to the left heart the pulsation may manifest itself in these spaces with greater or less intensity as far outwards as the axillary line, or when the right half of the organ is affected it may extend in the other direction to near the right mammillary line.

It is to be borne in mind, however, that the heart may pulsate in at least two intercostal spaces and yet show no trace of hypertrophy, as when it lies in immediate contact with a larger part of the chest-wall than usual, from retraction of the anterior border of the left lung as the result of atrophy. But never, in such cases, is the apex-beat felt to the outside of the mammillary line to the left or beyond the parasternal line to the right, a fact which, apart from other points brought out by further examination, renders it impossible to mistake this condition for a genuine hypertrophy of the heart.—Hypertrophy, especially in children and young persons, announces itself not only in the character of the impulse, but also by giving rise to a certain degree of *prominence* of the præcordial region; in older patients, whose chest-walls are more rigid, this prominence is more seldom observed.

The various signs which have been discussed in this section always enable us to recognise whether the heart is normal with regard to position, whether it is hypertrophied and dilated, whether the increase in size is limited to the left or right ventricle or affects both; and the diagnosis so arrived at is almost invariably a sound one, particularly when the examination of the impulse by inspection and palpation is followed up by the inspection of the arteries (see p. 223); but a moderate amount of hypertrophy of the *right* ventricle may escape notice by inspection when the heart is covered by emphysematous lung.

SYSTOLIC PULSATIONS,

(proceeding partly from the heart and partly from the great vessels).

1. *Systolic Pulsation in the Epigastrium.*

This may be synchronous with the apex-beat in the normal situation, or may exist alone, when no other impulse is perceptible in the præcordial region.—The first condition is very frequently observed when the heart is acting with increased energy, and is then simply owing to the transmission of the normal impulse to that part; it disappears when the heart again begins to act quietly.—In another class of cases the *epigastric pulsation* accompanying the normal apex-beat is merely the transmitted *pulse of the abdominal aorta.* In such circumstances it depends on the presence of conditions which either strengthen the pulse of the abdominal aorta or which facilitate its conduction to a distance; the force with which that vessel expands is increased in hypertrophy of the left ventricle from whatever cause arising (excepting only that connected with stenosis of the aortic orifice), and the impulse is more readily conveyed through thin and lax abdominal coverings (as in women after several pregnancies) and through an enlarged and depressed left lobe of the liver.—Epigastric pulsation propagated from the abdominal aorta appears an instant later than the heart's impulse, and is also not strictly confined to the epigastrium, but extends over a considerable part of the abdominal surface; in cases of aneurism of the abdominal aorta and of the cæliac artery the greater part of the abdominal wall may be caused to pulsate.—The recognition of epigastric pulsation conducted from the abdominal aorta is thus comparatively easy, as that vessel may be directly examined by the hand, provided the tension of the recti muscles do not interfere to any great extent with the proper performance of palpation. By compressing the abdominal aorta at a more distant point the epigastric pulsation may be increased in force.

The second variety of systolic *pulsation in the epigastrium*, that in which the heart's impulse is either entirely wanting in the normal situation or is weak and diffuse, occurs when the *diaphragm* is *depressed*, particularly when at the same time

1. Those systolic *retractions* which *accompany* the normal apex-beat appear in the third and fourth intercostal spaces; the heart is sometimes in these cases in all respects normal, generally it is acting unusually energetically, most commonly it is hypertrophied, and in contact with a correspondingly large portion of the chest-wall, the margin of the left lung being pushed back; such depressions are more frequent in young persons, particularly in children, whose thoracic parietes are thin, than among adults, whose thorax is less yielding and is protected by thicker coverings. In these circumstances, when the heart acts with undue force each systole is attended by an exceedingly rapid undulatory movement passing over the præcordial region from above downwards, and a slight recession of the surface in the third and fourth intercostal spaces. The explanation of these phenomena is very probably this, that with the systolic locomotion of the *apex* to the left, downwards and *forwards*, is combined a *movement of the base of the heart backwards* and to the right; this creates an empty space between the base of the heart and the chest-wall, to fill up which the superficial soft parts are dragged inwards. These depressions have no diagnostic importance.

This explanation of the occurrence of systolic recession in the intercostal spaces at a point situated above that at which the apex-beat is seen, is, however, not perfectly satisfactory; the objection to it which has most weight is one suggested by Friedreich, that during the systolic movement of the apex downwards the space left vacant by the contracting portion of the heart, to occupy which the tissues immediately over it are said to be retracted, is at once taken up by another part of the heart in a similar state of contraction. It is nevertheless quite conceivable that the upper segment of the cardiac muscle is less considerably enlarged, particularly in the direction of its antero-posterior diameter, than the lower portion, and that in that way the depression of the intercostal spaces may be brought about.

2. The less common form of *systolic retraction* is also the more important of the two, that in which the apex-beat is completely *wanting*. In these cases the soft parts in the fifth intercostal space recede, are not elevated as they should be, on each contraction of the heart; this change may either be limited to the area usually occupied by the normal apex-beat or may extend over nearly the whole of the præcordia, so that even the sternal ends of the ribs and the lower end of the sternum are drawn inwards. This phenomenon is pathognomonic of *adhesion* of the

heart to the pericardium (Skoda) or of the pericardium to the substernal connective tissue and the diaphragm. The degree and extent of the adhesion may be inferred from the depth and area of the depression: thus, if the heart be adherent to the pericardium and the vertebral column on one side, and to the sternum on the other, its movement downwards and forwards on contraction is prevented, and as all the diameters of the heart are shortened during the systole, and as the spinal column is a fixed point, the lower end of the sternum is retracted; if adhesions have also been formed with the connective tissue investing the ribs, the latter also are similarly acted upon; and finally, if the heart be completely adherent to the tissues round it on every side, the systolic depression involves a large part of the surface of the left lower segment of the thorax. To overcome all these difficulties in the way of the proper performance of its functions, the heart necessarily acts with increased energy.—It is nevertheless sometimes observed that depressions limited to the region in which the apex-beat usually manifests itself, may be dependent on adhesions of very trifling extent.*—When the contractile power of the heart is greatly diminished, the systolic retractions disappear, notwithstanding the existence of extensive and intimate adhesions.†

The parts of the thorax which, on account of the adhesions formed by the heart, are at each systole dragged inwards towards the vertebral column spring back again during the diastole to their former position; if the systolic retraction is confined to the spot at which, in normal circumstances, the apex-beat is felt, this region becomes markedly prominent during the diastole, constituting a *diastolic* cardiac impulse or apex-beat,—the only exception to the general rule that the apex-beat is systolic in rhythm.

INSPECTION OF THE ARTERIES.

The increased pulsation of the aorta in hypertrophy of the left

heart and in cases of aneurism has already been under consideration. In the same way the force of the pulsation in the further ramifications of the aorta,—the carotid and subclavian arteries, &c., may be taken as a measure of the degree of power being exercised by the heart.

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* Traube has described one case of distinct *systolic retraction*, in which there was *no adhesion* of the heart to the pericardium. The retraction was found, on *post mortem* examination, to be caused by the presence of a congenital fold on the posterior surface of the heart, passing from the upper end of the pulmonary artery to the left auricle, so situated that the systolic movement of the heart to the left and downwards was prevented, or at least limited.

† A case of this kind has come under my own notice: on examining the body of a patient who had died in the stage of asphyxia in cholera, the heart was discovered to be adherent at nearly every point to the surrounding tissues, yet during life no trace of retraction was seen (the cardiac impulse was not perceptible).

While the heart is acting quietly the arteries in the neck pulsate feebly, the carotid wave appearing only in the fossa between the sternomastoid muscles, and that of the brachial and radial arteries only when the arms are held in a suitable position. When the heart acts more powerfully, but still within physiological bounds, arterial pulsation is to a corresponding degree increased, and is particularly noticeable over the larger vessels. In hypertrophy of the left ventricle the pulse becomes still more forcible, that of the carotid being clearly seen through the soft tissues of the neck; and smaller vessels which at other times are scarcely visible, such as the temporal arteries and the smaller branches of the brachial and femoral arteries, pulsate prominently and appear *dilated* and *tortuous*.—The phenomena attendant on hypertrophy of the *right* side of the heart are characteristically different from those in the domain of the systemic vessels in enlargement of the left ventricle. Here the arterial pulse is not only not rendered stronger, but it is frequently feebler than usual, as, from the engorged condition of the pulmonary circulation, of which the hypertrophy of the right ventricle is but another of the results, the aortic system of vessels contains a smaller volume of blood than normally. Enlargements of the right and left ventricles are therefore often distinguishable from each other at the first glance by simple inspection of the arteries. And further, as any considerable degree of hypertrophy of the *left* ventricle, if the strongly pulsating arteries are at the same time dilated and tortuous, is generally due to insufficiency of the *aortic* valves, and as, on the other hand, hypertrophy of the right ventricle is most commonly caused by *mitral* lesion, a careful inspection of the arteries often enables us to come to at least a *general* conclusion regarding the seat of the impediment to the circulation.

INSPECTION OF THE VEINS.

The pathological phenomena observed on inspection of the s are

1. *Engorgement* of the venous system, and therefore visible dilatation of the superficial veins;
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VENOUS CONGESTION.

Of all the superficial vessels near the heart the cervical veins show the greatest differences with regard to the amount of blood they contain at different times in health and disease; they are therefore well adapted for examination by inspection. Even in normal conditions the external jugular vein as it crosses the sternomastoid muscle may, by simply turning the head to one side, be made to stand out prominently on the skin as a thin, somewhat bluish-coloured cord; the internal jugular vein, on the contrary, placed deeply as it is between the two lower divisions of the same muscle, never comes into view.—The reason for this is found in the fact that the vertical course of the cervical veins promotes a rapid flow of blood from the head towards the heart; this tendency is still further favoured by the act of inspiration, which lessens the pressure within the innominate veins and the superior vena cava (into which the jugular veins empty themselves), and in that way, by a kind of aspiratory action, quickens the current through these vessels.

As a pathological phenomenon congestion of *all* the veins of the body, particularly of those of the neck, is met with under the following circumstances:

1. When the contractile power of the right ventricle is diminished: the latter is then unable to discharge itself of the whole of its contents, and so fails to accommodate all the blood which should enter it from the right auricle, and the auricle itself, and eventually also the venous trunks and the whole venous system, become unduly distended with blood.—Of all the changes in the organs of circulation, mitral insufficiency and constriction of the left auriculo-ventricular orifice, and of the affections of the respiratory apparatus those which lead to persistent congestion of the pulmonary circulation, and so to hypertrophy of the right heart, and at a later stage to fatty degeneration of the cardiac muscular fibre, are the diseases which most commonly give rise

heart and in cases of aneurism has already been under consideration. In the same way the force of the pulsation in the further ramifications of the aorta,—the carotid and subclavian arteries, &c., may be taken as a measure of the degree of power being exercised by the heart.

While the heart is acting quietly the arteries in the neck pulsate feebly, the carotid wave appearing only in the fossa between the sternomastoid muscles, and that of the brachial and radial arteries only when the arms are held in a suitable position. When the heart acts more powerfully, but still within physiological bounds, arterial pulsation is to a corresponding degree increased, and is particularly noticeable over the larger vessels. In hypertrophy of the left ventricle the pulse becomes still more forcible, that of the carotid being clearly seen through the soft tissues of the neck; and smaller vessels which at other times are scarcely visible, such as the temporal arteries and the smaller branches of the brachial and femoral arteries, pulsate prominently and appear *dilated* and *tortuous*.—The phenomena attendant on hypertrophy of the *right* side of the heart are characteristically different from those in the domain of the systemic vessels in enlargement of the left ventricle. Here the arterial pulse is not only not rendered stronger, but it is frequently feebler than usual, as, from the engorged condition of the pulmonary circulation, of which the hypertrophy of the right ventricle is but another of the results, the aortic system of vessels contains a smaller volume of blood than normally. Enlargements of the right and left ventricles are therefore often distinguishable from each other at the first glance by simple inspection of the arteries. And further, as any considerable degree of hypertrophy of the *left* ventricle, if the strongly pulsating arteries are at the same time dilated and tortuous, is generally due to insufficiency of the *aortic* valves, and as, on the other hand, hypertrophy of the right ventricle is most commonly caused by *mitral* lesion, a careful inspection of the arteries often enables us to come to at least a *general* conclusion regarding the seat of the impediment to the circulation.

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to swelling of the cervical veins in the manner described; a good instance of such a sequence of events is furnished by chronic vesicular emphysema of the lungs.

2. When the pressure on the venous trunks within the thorax (the venæ cavæ) is from pathological causes so augmented that they are no longer able to receive all the blood flowing towards them.

As in normal conditions the increase of the intrathoracic pressure which takes place during expiration checks the flow of the venous current,—an effect, however, which is neutralized and even more than compensated for by the accelerating influence of inspiration, and which, therefore, in health is never seen externally,—so also in pathological circumstances a persistent increase of the same intrathoracic pressure results in an equally persistent overloading of the veins of the neck; copious pericardial and pleuritic effusions, large mediastinal tumours and aneurisms of the aorta, pneumothorax, and the severer forms of pulmonary emphysema (in which expiration is much prolonged and usually effected only with the aid of the auxiliary muscles of respiration), are among the affections in which the phenomena under discussion are observed. In the more advanced stages of emphysema the swelling of the cervical veins (and also of those of the body generally) becomes a very striking symptom, because not only does the right ventricle act more feebly, as already pointed out, but the pulmonary capillaries are extensively atrophied and obliterated and the volume of blood permitted to circulate through the lungs thus greatly reduced.

In rare cases, particularly of fibrous mediastinitis, the reverse of these appearances may be seen, distension of the jugular veins with each inspiration: the fibrous cords developed in this affection exercise a certain amount of traction on the intrathoracic veins each time the chest is expanded in inspiration, and in that way lessen the lumen of the vessels in question (compare p. 244).—Congestion may also be limited to well-defined districts drained by particular veins, when it is probably due to some *local* cause, such as thrombus, obliteration, or compression of one of the larger venous trunks; of this nature are certain of the congestions of the veins of the neck, from compression by large goîtrous tumours or swollen cervical glands.

The swelling of the veins of the neck is much more conspicuous while the patient is lying on his back than when standing or sitting, as in the former position the flow of blood into the auricle is considerably retarded. The *respiratory movements*, also, have an important influence on the cervical veins with respect to their state of distension, an influence which is, further, more marked when decubitus is dorsal; in inspiration the veins tend to collapse, the blood they contain being rapidly drawn off, while in expiration they are speedily refilled and stand out on the surface as distinct bluish cords. Coughing renders the veins still more turgid, the sinus of the jugular vein then becoming especially prominent and appearing as a large bluish-coloured swelling. These effects of the different phases of respiration, however, are noticeable only in those pathological conditions in which the jugular veins are persistently dilated and overfilled. In the healthy subject engorgement of the veins of the neck (or of the facial veins, causing cyanotic discoloration) may be excited by repeated complete expiration, by fixing and for some time retaining the chest-walls in the position most favourable to expiration, or by prolonged and severe coughing.

Movements in the Veins of the Neck.

These consist occasionally of a rhythmical *dilatation* of the veins, determined by the movements of respiration; or they may be of an *undulating* character, taking the form of a continuous, but non-rhythmical succession of waves passing along the distended vessel, due jointly to the expansion and contraction of the chest in respiration and to the action of the heart; or, finally, they may be of the nature of a *pulsation*, dependent solely on the movements of the heart (venous pulse).

Undulation of the Jugular Vein.

Undulatory movements, like the various degrees of congestion of the jugular veins coinciding with the different phases of respiration, indicate a state of engorgement of the pulmonary circulation. They, however, are also considerably modified by the action of the heart: an obstructed lesser circulation always leads to overloading of the right auricle, each systole of which throws a small quantity of blood back into the vena cava superior, engorgement of the latter ensues, the current of blood through the jugular vein becomes slower (or is possibly sometimes arrested for an instant), and local congestion is developed. This mass of blood thus forced backwards into the vena cava by the contraction of the over-filled right auricle, on the one hand, and the simultaneous retardation of the outflow of blood from the jugular veins, on the other, combine to dilate these veins and communicate a distinct impulse to their contents; and as the veins once thrown into commotion do not immediately return to a state of quiescence, and as they are further also subjected to some disturbance and caused to swell up by the expiratory movements, the agitation of their walls, when the impediment to circulation through the lungs is of a serious character, becomes *undulatory* and almost continuous.

Undulation of the veins of the neck, therefore, which occasionally also extends to the smaller vessels opening into the jugulars, can arise only in those cases in which the cervical veins show a considerable amount of engorgement even during inspiration, when circumstances are most favourable to the rapid passage of the venous current.—*Expiratory distension* (not undulation) is observed in the superficial *brachial veins*, but only during severe attacks of coughing.—Of all the diseases which involve congestion of the veins of the neck it is in intense pulmonary emphysema and in stenosis of the left auriculo-ventricular orifice that undulation of these vessels occurs most frequently and in its most marked forms.

VENOUS PULSATION.

This presents itself as a rhythmical *elevation* of the walls of the *internal jugular vein,* coinciding with the systole of the heart or sometimes barely preceding it, always clearly perceptible to sight and touch,—most clearly when decubitus is dorsal. The vein pulsates either in its whole length, the impulse being always propagated from below upwards, or only its lower part, the sinus, partakes in the movement.

The venous pulse frequently consists of only a *single* throb, synchronous with the systole of the heart; at other times the wave is *double*, in which case the first part, that preceding the systole (presystolic), is the weaker, and the second, that coinciding with the systole, the stronger. When the systolic pulse is double the diastolic collapse of the veins is divided similarly into two distinct movements. A double systolic wave (Anadicrotism) and a double diastolic subsidence of the walls of the vessel (Katadicrotism) are usually readily detected by simple inspection and palpation; the reduplication may be more accurately demonstrated, however, by means of the sphygmograph.

The persistent distension to which a pulsating jugular vein is subjected largely increases its calibre: in one case that was under my own care the breadth of the vessel was 1 ctm.—To mistake the venous pulse for that of the carotid artery is hardly possible; if it be suspected that the pulsation is *communicated* from that artery to the jugular vein such a source of error may easily be excluded by so altering the position of the head that these vessels are brought into less close relation to each other. If the pulsation be presystolic as well systolic, and somewhat feeble, it rather closely resembles simple undulation, from which, nevertheless, it may be at once distinguished by pressing upon the vein with the finger: *undulation disappears below the point at which pressure is made,* (that is, in the central part of the jugular vein, that nearest the heart), *while pulsation continues,* and is even intensified, by the proceeding described.

Venous pulsation is caused by the regurgitation of a wave of blood which, during the systole of the heart, passes upwards into the vena cava superior and through it into the innominate veins and the jugulars. Such regurgitation most commonly depends on insufficiency of the tricuspid valves; each contraction of the right ventricle drives a portion of its contents backwards through the right auricle into the superior cava and the innominate and jugular veins. The greater part of this backward wave reaches the *right* jugular vein through the *right* innominate vein, as the latter is continuous, in almost a direct line, with the vena cava superior, while the left innominate vein joins it at nearly a right angle. The venous pulse is therefore *more marked on the right side* than on the left; in the latter situation it may even be completely wanting.—In the early stages of the affection the upward progress of the impulse is checked by the closure of the valves with which the jugular veins are furnished, and *the pulse is thus confined to the lower part of the vessels, particularly to the sinus,* and coincides exactly with the systole of the heart; beyond the pulsating sinus *undulation* of the vein may be observed, the result partly of the shock which is transmitted through the whole length of the vein, and partly of the obstruction presented to the free outflow of blood. Gradually, however, the jugular valves are overcome by the persistent pressure from below, they lose their elasticity or are perforated or torn, and so become incompetent; the regurgitating wave then rises above the valves, and pulsation is perceptible in the jugular vein as high even as the angle of the jaw. In cases of insufficiency of the tricuspid valve, besides the systolic pulsation just mentioned, there may be a distinct, though much feebler, presystolic pulsation, due to the regurgitation of blood which takes place on contraction of the overfilled right auricle. Presystolic and systolic pulsations of the jugular veins are thus always, without exception, pathognomonic of absolute or relative insufficiency of the tricuspid valve.

Absolute insufficiency of the tricuspid valve is the result of organic lesion, while *relative* insufficiency is caused by excessive dilatation of the right auriculo-ventricular orifice from the unduly large quantity of blood contained in the right heart, the valves themselves being intact. Relative insufficiency of the tricuspid valve occurs also with stenosis of the mitral orifice*: venous pulsation is set up therefore in these cases, the whole length of the jugular vein being involved when its valves are incompetent, or only its lower part so long as these structures continue to perform their office.—But venous pulsation may also be observed, independently of the existence of any relative insufficiency of the tricuspid valve, in cases of stenosis of the mitral orifice; this symptom, indeed, is comparatively common in the severer forms of this valvular lesion. It is produced by the contraction of the overloaded right auricle, (and is accordingly presystolic), is but feeble, and limited to the lower part of the jugular vein.—And finally, in certain rare cases venous pulse is developed in the absence of any affection of the heart, that is, without mitral lesion, from simple uncomplicated insufficiency of the valves of the jugular vein (Friedreich). In pathological conditions, (pulmonary emphysema for example), which give rise to persistent obstruction of the current of blood through these vessels, their valves yield gradually to the strain they have to bear in each expiration, they become relatively or really insufficient, and on the contraction of the right auricle a quantity of blood is thrown back into the jugular vein; but a venous pulse generated in this way is necessarily very feeble.

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* This may occasionally arise even though the mitral valve be structurally unaffected, as in a case which I recently had an opportunity of observing, in which a considerable dilatation of the right ventricle appeared as one of the results of pulmonary phthisis; on *post mortem* examination the tricuspid valve was found to be normal, the orifice, however, being wide enough to admit three fingers, while the mitral valve and orifice showed no trace of morbid alteration. During life a systolic murmur was audible at the lower part of the sternum, and the extremely dilated jugular veins pulsated synchronously with the systole of the heart.

In uncomplicated cases sufficient evidence is usually furnished by simple inspection of the venous pulse to enable us to tell whether it is caused by incompetency of the tricuspid valve, or, the latter being normal, by insufficiency of the valves of the jugular vein from prolonged distension of the right auricle: in the former case the throb is distinct, forcible, and systolic, in the latter case it is always presystolic; auscultation of the heart also shows that tricuspid insufficiency is marked by a loud systolic murmur, audible even in the jugular vein, while if the valvular apparatus in question be intact only a pure systolic heart-sound is heard. Whether, on the other hand, the pulsation arises from a real or only a relative incompetency of the tricuspid valve, can not be made out with confidence in every case, as in both conditions, in addition to the similarity of the appearances so far as the pulse itself is concerned, there is the same systolic murmur over the right heart, though in absolute insufficiency it is much louder than when the defect is merely relative. If in the later stages of tricuspid insufficiency the contractile power of the right ventricle is diminished the venous pulsation becomes feebler, and may even disappear entirely if the backward impulse do not travel so far peripherally as the jugular veins.

But it is not possible by means of inspection alone to determine with certainty in every case whether the pulsation is limited to the sinus of the jugular vein or extends also to a further point in the course of the vessel, a variable amount of undulation being generally added to the pulsatory movement, as the vein when agitated does not at once return to a state of rest. In such circumstances palpation offers more reliable information: thus, if the finger be placed on the pulsating jugular vein at the parts at which it is provided with valves there is felt a systolic thrill, which owes its existence to the regurgitation of blood through the incompetent valves, when the heart's action is sufficiently strong, and particularly during an attack of coughing. This phenomenon is naturally also appreciable to the ear as a more or less loud murmur. If, on the contrary, the jugular valves be competent this fremitus is wanting, and is often replaced by a clearer sound, the sound of the jugular valves (Bamberger), produced by the sudden tension of these parts from the impact of the regurgitant wave.

In general the retrograde impulse does not go beyond the internal jugular vein; in other cases, however, smaller vessels which join it also pulsate, relatively most frequently the external jugular and thyroid veins, more seldom the veins of the face. Should a portion of the wave pass from the innominate into the subclavian veins, the larger veins of the regions drained by these venous trunks, the axillary, brachial, and the superficial veins of the arms, exhibit similar pulsatory movements.

Occasionally the pulsation is propagated not only through the vena cava superior but also into the *inferior cava;* but it appears in the latter vessel and in those discharging into it, only when the force of the impulse is sufficiently great, and that occurs only in cases of absolute insufficiency of the tricuspid valve. If the wave be feeble it is lost before it penetrates any distance into the vena cava inferior. That a venous pulse should be so rare in the domain of the inferior cava is explained partly by the fact that the situation of the orifice by which that vessel opens into the right auricle is very unfavourable to the admission of a backward current of blood, and partly by the great length of the course of the vein before it becomes accessible to palpation in the abdominal cavity.

Not unfrequently the impulse traverses the vena cava inferior, reaching as far as the *hepatic veins,* when it gives rise to a rhythmical pulsation of the liver, following closely after the stroke of the heart. If the liver be also increased in size, as in such cases it generally is, from the congested state of all its veins, so as to form a more or less prominent tumour below the ribs, this pulsation is perceptible to the finger applied over any part of the organ, and is thus easily distinguished from the circumscribed elevation of the left lobe of the liver which is so often caused by the systolic expansion of the subjacent abdominal aorta.

Hepatic pulsation in cases of insufficiency of the tricuspid valve is partly, though not exclusively, due to the rhythmical dilatation of the vena cava inferior, which lies behind the liver: apart from the fact that the pulse of this vessel is too feeble to raise such a heavy organ to any marked extent, the feeling communicated to the hand is not merely that of uplifting of the liver as a whole, but at all points it is of the nature of a pulsation.—In certain cases the pulse of the hepatic veins is an earlier phenomenon than that of the jugular, the inferior cava not being provided with any valvular apparatus which might for a time stem the backward current of blood through it to the veins of the liver (Friedreich).

Pulsation of the femoral veins is sometimes, (though very rarely), observed as a result of insufficiency of the tricuspid valve, as the greater part of the blood regurgitating into the vena cava inferior enters the hepatic veins, only a very small portion of it going past the orifices of these vessels.

It may be here remarked that feeble *pulsation* sometimes also occurs as a *physiological* sign in the superficial *veins on the back of the* *hand and the dorsum of the foot*, when the impulse generated by the contraction of the left heart is not abolished in the capillaries, but passes through the latter (giving rise to a capillary pulse, visible in the finger nails) into the veins. Quincke has directed special attention to this symptom, which, however, had been noticed by several earlier authors. It may appear in those who are in perfect health, but is most common in cases in which the pressure within the arteries is pathologically increased and suddenly diminished,—particularly, therefore, in aortic insufficiency, but also, as Quincke, and more recently Peter and Broadbent have shown, in various other conditions characterised by a certain degree of relaxation of the arteries.

PALPATION OF THE PRÆCORDIAL REGION.

THE investigation (by means of palpation) of the phenomena which depend for their existence on the action of the heart, and which manifest themselves in the præcordial region and the parts in its vicinity, is in many respects supplementary to inspection, the results already acquired by the latter method being completed and rendered more intelligible by those obtained by the former, though palpation alone is also capable of affording very important information. Very frequently palpation and inspection are employed simultaneously, a fact the practical bearing of which has been repeatedly pointed out while discussing the signs observed in inspection.

The phenomena to which examination by palpation is usually directed are: the *cardiac impulse*, its force and extent; *pulsations* appearing at various points on the surface of the thorax, and connected directly or indirectly with the impulse of the heart; *murmurs* which communicate a certain feeling of vibration to the *hand* in the præcordial region; the *arterial pulse*.

Palpation of the *impulse of the heart* yields substantially the same indications as inspection, but with more rigorous exactness, particularly as regards its situation, extent, and force. Slight increase of the energy with which the heart contracts, an impulse of a heaving character, extension of the area of impulse towards the right, &c., are conditions the presence of which can often be determined with certainty only by the aid of palpation.

Murmurs associated with tactile vibration are occasionally felt in the præcordial region, coincident with the heart's contraction (systolic), going immediately before it (presystolic), following closely after it (diastolic), or occurring irregularly in the pause. Systolic, diastolic, and presystolic thrills of this kind originate *within* the heart or at the root of the great vessels, while those observed in the interval between the contractions of the heart arise in parts *external* to that organ; the former are termed *endocardial* thrills, the latter exocardial, or, as they are generated within the pericardium, *pericardial* thrills.

ENDOCARDIAL THRILL.

Endocardial thrill gives to the hand the impression of light rubbing or vibration, a sensation which so resembles the purring of a cat that Lænnec bestowed on it the name *fremissement cataire*. It is set up when the blood-stream during the systole or diastole or (rarely) during both phases of the heart's action, is thrown into vibration, acquiring a whirling motion, from contact with degenerated *valves*, or at contracted valvular orifices, or in pathological dilatation of the *initial portions of the great vessels. This vibration, which is felt as a thrill, is also audible as a murmur;* but every such disturbance within the organs of circulation is not sufficiently great to make itself sensible externally to palpation, only a comparatively small proportion of audible murmurs being associated with tactile vibration. Endocardial thrill usually increases in intensity as the heart's action becomes more energetic, or may even in that way be first called into existence.

1. SYSTOLIC THRILL.

This may be developed at the mitral, tricuspid, aortic, or pulmonary orifice. It is either most marked at the point at which it originates, provided there be no obstacle to its transmission to the surface at that situation,—as when the heart is covered by lung,—or it is propagated most distinctly in the direction of the current of the blood.

Purring tremor, systolic in rhythm, felt most intensely at the apex and becoming feebler the further the hand is removed from that part, either to the right or upwards, is invariably due to insufficiency of the mitral valve. Immediately over the valve itself (at the sternal insertion of the third rib, in the second left intercostal space) it is not appreciable, the base of the heart being there sheltered behind a process of lung tissue.

Systolic vibration in insufficiency of the mitral valve is nevertheless far from common, occurring, according to my observations, which include nearly 200 instances of this valvular lesion, in little more than a fifth of the cases; it may be wanting even though the accompanying murmur be loud and harsh.

Systolic fremissement most perceptible at the lower end of the sternum is generally transmitted from the aortic orifice, or is caused, in very rare cases, by incompetency of the tricuspid valve.

Such a thrill is to be referred to incompetency of the tricuspid valve only when associated with pulsation of the jugular veins. In four cases of tricuspid insufficiency which came under my notice there was no trace of vibration of this kind.—Most commonly it is *propagated* from the aortic orifice, in cases of stenosis or atheromatous degeneration of the vessel, and in these circumstances is diffused over a large part of the sternum and the adjoining costal cartilages.

A systolic thrill *confined* to a spot in the second left intercostal space, near the sternum, may arise from roughness or contraction of the pulmonary orifice, both very rare valvular affections; it may also be due to mitral insufficiency, when, for instance, the base of the heart lies in immediate contact with the chest-wall, from retraction of the anterior margin of the left lung.

Systolic *fremissement* in the second right intercostal space, close to the sternum, radiating along the body of the bone even to the ensiform process and to the adjacent insertions of the ribs on the right side, proceeds from roughness or contraction of the aortic orifice, from atheromatous degeneration of the aortic walls, and from aneurisms of the ascending aorta.

2. Diastolic Thrill.

Diastolic thrill most frequently takes its rise at the mitral orifice, more rarely at the aortic orifice, still more seldom at the pulmonary orifice, and is scarcely ever dependent on uncomplicated disorder of the tricuspid orifice.

Constriction of the *mitral orifice* produces a diastolic thrill, the current of blood being agitated or thrown into eddies in passing from the left auricle into the left ventricle. This tremulous sensation is felt most distinctly at the apex of the heart, is usually spread over a somewhat large area, but is not at all points of equal intensity. It exists in every case of *considerable* stenosis of the left auriculo-ventricular orifice, or, if it be wanting, may usually be developed by whatever tends to increase the heart's action,—raising the arms rapidly, or walking quickly to and fro, &c. It lasts either throughout the whole of the diastole, or appears only at the end of it, shortly before the systole, and is therefore sometimes also called *presystolic* thrill. If it be prolonged through the whole diastole to the next systole it is generally feebler at its commencement; at the end of the diastole, that is in the presystole, it is suddenly much augmented in intensity by the contraction of the auricle, which presses the blood with greater force through the narrow mitral orifice. Such cases are usually also characterised by a loud murmur, quite perceptible to the hand, but still more so to the ear, similarly divided into two stages or portions (see Mitral Murmurs).

The diastolic thrill generated at the *aortic orifice* does not, like that originating at the mitral orifice, show the jerking quality just mentioned, but is continuous, of nearly equal intensity during the whole of the diastole. It occurs, though not very frequently, in insufficiency of the aortic valves, and is owing to the regurgitation of blood from the aorta into the left ventricle. It may usually be felt over the whole of the sternum, but is most fully developed at that part behind which the aortic orifice really lies. *Fremissement* of this description, radiating to some distance over the sternum and further also to the left or right, appears not only in aortic insufficiency but also as one of the signs of large aortic aneurisms.

Diastolic thrill from valvular lesion at the orifices of the *right* heart is exceedingly rare.

Diastolic thrill over the *pulmonary orifice* is connected with incompetency of the pulmonary valves; it manifests itself in the second left intercostal space at the sternal insertion of the third rib, and is limited to a comparatively small area.

On only one occasion have I felt a diastolic thrill clearly traceable to pulmonary insufficiency. Its situation, and the point at which it is of maximum intensity, effectually guard against its being confounded with a thrill from any other source. Such a mistake is possible only in those comparatively rare cases of mitral stenosis in which the left auricle, being exposed by the retraction of the anterior border of the left lung, comes into close contact with the thoracic parietes, when a similar thrill is noticed also in the second left intercostal space. In these circumstances, however, the tremor is strongest at the apex of the heart, and is further diffused over a much larger surface than the vibration due simply to pulmonary insufficiency.

Diastolic thrill originating at the *tricuspid orifice* is always the result of stenosis. This lesion is one of the rarest in the whole range of heart affections, and almost never occurs without complication.

PERICARDIAL THRILL.

Pericardial friction-sound is frequently accompanied by a thrill, produced by the movements of the heart, the visceral and parietal layers of the pericardium, roughened by inflammatory fibrinous deposit, being thereby caused to rub on each other.—As this sign is better appreciable by the ear than by the hand, it is reserved for discussion in the chapter on Auscultation.

PULSATIONS OF THE LARGE VESSELS.

Palpation also proves useful in the investigation of certain circumscribed pulsations observed at various points on the anterior surface of the chest, and dependent on the movements of the great vessels, the aorta and subclavian artery. The causes of these pulsations (undue expansion of the vessels from hypertrophy of the left ventricle, or *aneurismal* dilatation of the aorta) have already been under consideration on p. 220, in the section devoted to Inspection.

Palpation elucidates their nature more fully and exactly than Inspection, particularly with regard to the area they involve, their force, and the presence or absence of thrill.

From the pulsations just mentioned, due to dilatation of the arteries and synchronous with the systole of the heart, certain others are to be distinguished, *diastolic* in rhythm, and confined to a small part of the great vessels close to their origin. There is thus sometimes seen and felt in the *second left* intercostal space close to the sternum a strictly circumscribed pulsation, appearing an instant later than the heart's impulse, that is, in the diastole; in very marked cases this alternate elevation of the tissues over the apex and those in the second intercostal space is well brought out by lightly placing a finger on each of the spots indicated. This short *diastolic* stroke proceeds from the pulmonary artery, and is connected with *hypertrophy of the right ventricle;* it is the expression of the *unduly forcible closure of the pulmonary valves*, from the abnormally energetic recoil of the blood within the vessel. The conveyance of this impulse to the chest-wall is favoured by conditions in which the rigidity of the thorax is diminished (it is therefore most perfect in children), and by *retraction of the anterior border of the left lung*, such as results from hypertrophy of the right ventricle, when no lung-tissue is interposed between the pulmonary artery and the thoracic parietes. Where there is no retraction of the lung the diastolic impulse of the pulmonary artery is exceedingly feeble or absolutely wanting; it disappears also at a later stage when the contractile power of the right ventricle is lessened by fatty degeneration of the muscular substance of the heart. Inasmuch as the pulsation in question can be set up only by a very considerable degree of hypertrophy of the right ventricle, and as such enlargement results only from defects of the mitral valve, a diastolic impulse plainly emanating from the pulmonary valves may be looked upon as an almost certain sign of mitral insufficiency or stenosis of the left auriculo-ventricular orifice.—This phenomenon presents itself also to the ear in the greater loudness of the pulmonary arterial sound.

More rarely a *diastolic impulse* is felt in the *second right* intercostal space, close to the sternum. It comes from the aorta, and is occasioned by the *abnormally forcible closure of the aortic valve, observed in hypertrophy of the left ventricle*. Its rarity is due to the fact that the conditions which give rise most frequently to hypertrophy of the left heart are precisely those in which the aortic valve is more or less diseased and incompetent; this form of pulsation therefore is limited to those cases of enlargement of the left ventricle which, unattended by aortic valvular lesion, depend on contraction of the kidneys or sclerosis of the arteries, though even in such circumstances the symptom is seldom met with, as the heart is not always sufficiently hypertrophied, nor does the aorta approach close enough to the chest-wall, to ensure the ready transmission of the diastolic valvular impulse.

Examination of the Arterial Pulse.

The pulse is always taken at the radial artery, or at the carotid or other superficial arteries (the brachial or femoral) for the sake of comparison.

1. Frequency of the Pulse.

The acceleration of the pulse in all febrile conditions has already been referred to (p. 8). In diseases of the heart the frequency of the pulse is most commonly increased, seldom normal or diminished. It is invariably increased in the acute affections of the heart (endocarditis, myocarditis, pericarditis), very generally (among the chronic diseases of the heart) by valvular lesions, and in all those disorders grouped together under the term cardiac neuroses.

The rate of the pulse in the chronic, apyrexial diseases of the heart (valvular lesions) varies from 80—120 beats per minute, the higher numbers being reached only occasionally, when the heart is stimulated to greater activity by physical exertion. The pulse of mitral lesion often differs very markedly, with respect to frequency, from that of aortic disease: in the former it is almost always rapid, in the latter it may be scarcely altered, normal, or even (as in cases of constriction of the aortic orifice) sub-normal. A slow pulse sometimes accompanies atheroma of the aorta, probably from mechanical irritation of the cardiac branches of the vagus.—Patients suffering from idiopathic fatty disease of the heart, also, have usually a remarkably slow pulse, (in two such cases noted by me it fell, in the one to 28, in the other to 22, per minute), though in some instances it is normal or even accelerated. The pulse may further be very strikingly retarded, numbering 18 or even fewer beats per minute, without any disease of the heart being objectively recognisable; causes located in the nervous centres may then be suspected, and now and then actually demonstrated.

2. Rhythm of the Pulse.

In health the strokes of the pulse follow each other at perfectly regular intervals, that is to say, the heart is rhythmical in action. This rhythm, however, is subject to very frequent disturbance: in the minor degrees of irregularity the arhythmical contractions of the heart are few and occur at variable points in a moderately long succession of rhythmical contractions; in the severer forms the pulse loses all trace of regularity, and its individual beats very generally become unequal in volume and force.—Absence of rhythm may appear in the course of the most widely different diseases, as the expression of a derangement of the innervation of the heart; slighter deviations in this direction are also witnessed in physiological conditions, especially in advanced old age, and transiently under psychical influences. The greatest amount of irregularity is found in cases of cardiac disease, particularly of valvular affections, and of these it is most frequently attendant on mitral lesion. Irregularity of the pulse is most commonly first developed in heart diseases at a rather advanced stage, when the compensatory structural changes fail to answer their purpose, the only exception to this rule being stenosis of the left auriculo-ventricular orifice, in which this symptom may make its appearance while compensation is still perfect.

An artificial and temporary disturbance of the rhythm of the pulse may be excited, in cardiac diseases, by the use of digitalis.

A peculiar variety of irregularity of the pulse has been described by Traube, under the designation *pulsus bigeminus.* This consists of a pulse in which the beats run in pairs, a somewhat protracted pause occurring regularly between every two pulsations, that is, between every two contractions of the heart. The cause of the bigeminate pulse has not yet been satisfactorily made out. This and other modifications of the rhythm of the pulse are sometimes discovered in animals in which the intracardiac blood-pressure is augmented. In men it is associated almost exclusively with the existence of some obstruction to the circulation (valvular defects, &c.).—Instances of pulsus *trigeminus*, in which every third beat is followed by a pause, have been put on record by Riegel, Rosenstein, and others.—A further variation of the bigeminate pulse is presented in the pulsus *alternans*, which is characterised by the regular alternation of a small feeble pulsation with one which is larger and stronger (Traube). Like the pulsus bigeminus it appears, according to Schreiber, in a great variety of conditions, relatively most frequently in cases of mitral lesion. Occasionally the second of the two beats becomes so feeble as to be no longer recognisable by the finger, notwithstanding the fact that both contractions of the heart are still distinctly appreciable to palpation in the præcordial region ; there is but *one* pulsation at the wrist, therefore, to represent every two contractions (Fräntzel), the second of which is thus *abortive*.—The irregularities of pulse just named merge readily into each other, each form then lasting for a variable period. Arhythmical action of the heart, particularly that which depends on the administration of digitalis or on mitral stenosis, is often for a short time accompanied by a pulse having the characters of the pulsus bigeminus or the pulsus alternans, but it is very seldom that these phenomena are repeated for any length of time with such a degree of constancy that the irregularity becomes really rhythmical in its recurrence.—Another and somewhat rare variety of pulse has been described, the *pulsus myurus*, in which a full and forcible pulse-wave is followed by a series of several beats gradually decreasing in volume, this succession of changes being maintained with a certain degree of regularity.

The rhythm of the pulse may further be disturbed in such a way that the blood waves do not arrive at the two wrists at precisely the same moment. This inequality is met with when the aorta is the seat of aneurismal swelling, circulation being delayed in the arteries of the side towards which the aneurism lies; it is most evident when the tumour is situated on the arch of the aorta, between the vessels which spring from it,—the innominate artery (or right subclavian) and left subclavian.

In other cases of aneurism, especially of the ascending aorta, the pulse in the radial arteries is not indeed unequal, but is considerably postponed; instead of occurring directly after the cardiac systole, it does not appear till a distinctly appreciable interval has elapsed. This is also sometimes caused by stenosis of the aortic orifice, from the longer duration of the systole in that affection. And in the severer forms of aortic insufficiency the pulse, more particularly that of the carotids, is not synchronous with the heart's impulse, but is felt an instant later, a circumstance which has been explained by the supposition that the blood projected into the aorta at the beginning of the systole encounters the regurgitating blood-stream, and is therefore later in reaching the arteries of the neck (Tripier).—Finally, large aneurisms of the descending aorta tend to retard the pulse-wave, so that it is not perceptible in the femoral arteries so soon as in the radials.

3. Intermission of the Pulse.

In observing the pulse it is often noted that after several regular strokes one or sometimes two are omitted. This depends on one of two conditions: either the heart's action is periodically interrupted (*pulsus deficiens*), which is most commonly the case, or more rarely certain of its contractions, though regular enough, are not sufficiently energetic to give rise to a corresponding throb of the radial arteries. Such an intermittent pulse is sometimes seen to be compatible with the enjoyment of perfect health in other respects; it presents itself also in very diverse affections, independently of any disease of the heart, though in a large number of cases it is associated with positive cardiac disorder. It is often connected with a partially-filled or shrunken state of the left ventricle (from mitral stenosis or mitral incompetency), when an abnormally small quantity of blood is thrown into the aortic arterial system on each contraction of the heart; it is most frequently due, however, to that diminution of the heart's contractile power which takes place in the later stages of every cardiac disease.

4. VOLUME OF THE PULSE.

The largeness or smallness of the pulse depends on that of the blood-wave which passes through the radial artery, and this again depends, *cæteris paribus*, on the capacity of the artery at the wrist. Even in health the circumference of these vessels is very different in different persons, some having wide arteries and a full pulse, others slender arteries and a small pulse; between those extremes numerous gradations are observed. The volume of the pulse in health and disease also varies with the energy of the heart's action, though even in the absence of any undue excitement of the organs of circulation many individuals present a pulse the strokes of which are very unequal. When the radial artery is permanently distended, as in insufficiency of the aortic valves, it becomes the seat of persistent pathological *dilatation*, and the pulse is in consequence *increased in volume*. On the other hand, diminution of the calibre of the artery and of the volume of the pulse takes place when the quantity of blood circulating in the systemic arteries is abnormally small, a condition which is realised in the following circumstances: in stenosis of the aortic orifice; in cases in which the descending aorta or the aortic arch is compressed by morbid growths, such as mediastinal tumours; when the arteries of the body generally are unduly contracted; in all diseases of the heart in which the systemic arteries are deprived of their proper share of the blood in circulation, from congestion of the pulmonary vessels and of the whole venous system—in mitral stenosis, mitral insufficiency, &c. The pulse always becomes smaller as the heart's action begins to fail: if the heart be very seriously enfeebled, as it is in the last stages of those affections which terminate fatally, and temporarily in ordinary cases of fainting, pulsation is so slight as to cause scarcely any appreciable elevation of the artery (*threaddy pulse*) or merely a slight quivering movement is communicated to the arterial wall (*tremulous pulse*). In the asphyxial stage of cholera the radial pulse disappears altogether.

Rhythmical variations are sometimes observed in the volume of the pulse, dependent on the influence of the respiratory movements; the characteristic features of this sign are that *simultaneously with each inspiration* the pulse-wave in all the arteries is *reduced* in magnitude, or is absolutely *suppressed* on making a full and deep inspiration, but returns to its normal volume during expiration. This form of pulse, first noticed by Griesinger, and named *pulsus paradoxus* by Kussmaul, occurs in various diseases, and is an index of the presence of one of two conditions: the existence during inspiration of a direct mechanical obstacle to the emptying of the left ventricle into the aorta, or increase of the negative inspiratory pressure from changes which prevent the free entrance of air into the lungs. The most striking examples of the first condition are found in *fibrous mediastinitis* and *fibrous pericarditis;* the firm cords of connective tissue developed in these affections, passing from the sternum and pericardium upwards and backwards towards the great vessels, enclosing the latter and to some extent dragging them from their normal position and attaching them more or less intimately to the sternum, are put on the stretch when the thorax is dilated in inspiration, and grasp and constrict the aorta, innominate artery, &c. Only a portion of the contents of the left ventricle, therefore, finds its way into the aorta during inspiration, the arterial pulse is rendered feebler, or even disappears if the chest be expanded to its utmost; in expiration, on the contrary, the aorta regains its natural calibre and the pulse its normal volume. It is plain also that the pulse will be full enough which responds to those contractions of the heart which do not happen to coincide with an inspiration.—But the *pulsus paradoxus* is also observed in certain cases of pericarditis in which the aorta is free from the embrace of such adhesions. Its occurrence in these circumstances is ascribed to various causes: to occasional embarrassment of the left ventricle during inspiration, from thickening of the pericardium associated with atrophy of the muscular structure of the heart (Traube, Stricker); or to the pressure of pericardial effusion, which retards the flow of blood from the venæ cavæ into the heart, particularly during expiration (when the intrathoracic tension is augmented), so that the heart contains less blood at the beginning of inspiration than at the commencement of expiration (Bäumler).—The second condition described as favourable to the production of the *pulsus paradoxus* is found in *stenosis of the air-passages.* Even in health the pulse may be slightly of this character, as each inspiration lessens the pressure within the thorax and within the aortic system of vessels generally, while expiration, on the contrary, adds to this pressure; the arterial pulsations therefore, which correspond to such contractions of the heart as are coincident with inspiration are smaller than those felt during expiration. These differences, though usually so trifling as to be inappreciable by the finger, are readily demonstrable on a sphygmographic tracing, the primary wave of the inspiratory pulsations being considerably lower than that of the expiratory pulse (Riegel). If, however, the inspiratory negative pressure within the thorax be abnormally low, as in cases of stenosis of the larger air-passages, and if to this be added as a second factor diminution of the contractile power of the heart, the inequalities just mentioned are much exaggerated, and are easily detected by the unaided sense of touch, pulsation being sometimes completely abolished during inspiration.

Inequality of the pulse in one radial artery as compared with that in the other (*pulsus differens*) may be of physiological or pathological origin. Of the former nature are those instances in which the vessel divides into its two branches at a higher point in its course on one side of the body than on the other, and so comes to be of comparatively small calibre at the part at which the pulse is usually taken; and as this anomaly is not on the whole very rare, it should always be looked for when marked difference in the radial pulses presents itself. Pathological inequalities of this kind are most commonly due to pressure on the arteries of one side, as by intrathoracic tumours implicating the aorta between the left common carotid and the left subclavian, occasionally also by tumours (carcinomatous lymphatic glands) which press upon the axillary artery; partial obliteration of one of the brachial arteries, and other local and generally easily recognised causes, often give rise to the same phenomena.

5. TENSION OF THE ARTERY.

By this term is meant the pressure exercised by the blood on the inner surface of the vessels. It reaches its maximum when the expansion of the artery is at its height, and sinks to its minimum at the end of the contraction of the arterial wall. The greater the distension of the vessel by the blood-wave the greater is its tension. An artery in a state of extreme tension is less compressible than one whose walls are more relaxed; the former is hard to the touch, the latter soft, a tense artery thus making what is technically known as a *hard pulse*, a relaxed artery a *soft* pulse. Other things being alike the intra-arterial pressure is increased by hypertrophy of the left ventricle, and diminished when the action of the heart is weakened, or when its cavities contain less than the normal amount of blood.—The degree of tension of the arterial wall during expansion determines also the extent to which the artery springs into prominence with each stroke of the heart; the harder and stronger the pulse the greater will be the elevation of the artery, provided that the capacity of the vessel is not in any way lessened, (as, for instance, in cases of stenosis of the aortic orifice).

Constrictions of the left venous and arterial orifices offer good examples of affections in which the differences in tension shown by the pulse are well-marked. In the former the left ventricle receives less than the normal volume of blood on which to expend its energy and consequently begins to atrophy at a comparatively early stage of the disease, when the pulse becomes smaller and of low tension; in stenosis of the aortic orifice, on the other hand, though the blood-wave is indeed small, as in the preceding case, and the radial arteries also contracted, the blood is forced onwards by all the power of a hypertrophied left ventricle and the tension of the pulse is accordingly considerably raised.

6. Celerity (activity) of the Pulse.

This term is used to express the relation which exists between the duration of the expansion and that of the contraction of the artery.

In the normal condition the expansion of the vessel by the wave propagated from the heart lasts almost exactly as long as its contraction; this, at least, is the impression given to the finger, but the sphygmographic tracing shows that the lines formed by the dilatation of the artery are really shorter than those corresponding to its elastic recoil. The relation of these different parts of the tracing to each other, however, is subject to great variation. If from pathological causes the artery attain at once its maximum of expansion, and return equally rapidly to a state of quiescence, a pulse is produced which is designated the *pulsus celer*,—a brisk, active pulse. The slighter manifestations of this peculiarity are scarcely appreciable by the finger, but in well-marked cases such a pulse is exceedingly characteristic, and is usually spoken of as a *bounding* pulse. This nimbleness in movement displayed by the arterial wall is the more readily recognised the larger the vessel in which it is observed; it is therefore more distinct in the brachial and femoral arteries than in the radials. The most exquisite examples of this symptom are found in aortic insufficiency: the arteries are quickly and forcibly dilated by the pressure of the blood driven into them by the enlarged and powerful left ventricle, but their recoil follows as speedily, the systolic wave being exhausted in two directions,—centrifugally (into the capillaries) and centripetally (by regurgitation into the ventricle). A sharply-defined pulse, therefore, is regarded as pathognomonic of insufficiency of the aortic valves.

A pulse distinguished by the opposite qualities is termed a *sluggish* pulse (*pulsus tardus*). It is noticed particularly when the arteries lose their elasticity, as from sclerosis. Vessels so affected present unusual resistance to expansion by the pulse-wave and subside equally slowly in the intervals; they feel firmer to the touch than in health, and in exaggerated cases may become so hard as to be absolutely incompressible.

7. Dicrotism of the Pulse.

Though the finger applied to the radial artery in the healthy subject is sensible of but one impulse, occurring during the period of expansion of the vessel, it may be demonstrated by means of the sphygmograph (see p. 248) that in the period of contraction also a slight elevation of the artery takes place,—usually indeed two such elevations are observable in the tracing. These are caused by recoil-waves, that is, by blood-waves which, during the contraction of the arteries, flow backwards towards the heart, where they encounter the closed aortic valves, rebound from these and again travel outwards towards the periphery. But if the tension of the arteries is lessened, as in high fever, and if, further, the primary impulse produced by the systole of the heart be short and vigorous, the reflected wave may effect such a considerable degree of diastolic elevation of the artery as will render the use of the sphygmograph superfluous, the unaided finger being sufficient for the detection of the feebler *after-stroke*. This is designated the *double*, or *dicrotic* pulse. It is fully developed only when the temperature becomes decidedly febrile, 39°—40° C. (102·2°—104° F.), particularly when it remains for some time at this point, as in typhus and other affections characterised by continued fever. In individuals already exhausted and emaciated by chronic disease very slight fever and a comparatively low temperature suffice to give rise to distinct dicrotism of the pulse, as the arterial tension in such patients gives way more rapidly than in cases of acute disease in previously healthy persons. In febrile attacks of short duration, also, as in intermittent fever, dicrotism is observed, occasionally even when the pulse is not at all increased in frequency; this fact is direct proof that the high temperature, apart from the other phenomena of fever, is of itself to be regarded as the cause of dicrotism (Riegel).

In those who are much emaciated, in whom the radials pulsate prominently, a double pulse may be recognised, without applying the finger to the wrist or having recourse to the sphygmograph, by examining the artery in powerful sunlight, when it will be seen that each primary pulsation casts a shadow, which is followed by a second shorter shadow, on the adjacent part of the surface. Dicrotism sometimes also manifests itself in the double jerking movement occasioned by the pulsation of the popliteal artery and communicated to the dependent leg and foot when the patient sits with one knee crossed over the other. In large animals this reduplication of the pulse may be demonstrated by puncturing the femoral artery and causing the stream of blood issuing from it to impinge on a strip of paper moving slowly past it, on which it forms a tracing; this process, described by Landois, is termed *Hæmautography*.

The *hyper-dicrotous* pulse is observed in very high fever, in which the temperature ranges from 40°—41° C. (104°—105·8° F.); in it the second beat is somewhat later than in the ordinary dicrotous pulse, appearing immediately before the renewed expansion of the artery, so that it closely resembles a preliminary systolic stroke. It is, however, really a dicrotic pulse, the peculiar character of which is due solely to its rapidity, each primary pulsation following so hard upon the preceding secondary stroke that the latter is shortened and to a certain extent rendered abortive. If the pulse become still more rapid even this shortened after-beat (or preliminary pulsation) disappears, and the dicrotic pulse merges into one which is strictly monocrotic.—A pulse which is obscurely dicrotic is named *sub-dicrotic;* it is noticed in moderate fever, when the temperature is only slightly elevated.

SPHYGMOGRAPHY.

(The graphic representation of the arterial pulse.)

The undulations of the pulse in all the superficial arteries may be measured instrumentally and represented graphically in tracings. The apparatus which is almost universally employed for this purpose is that invented by Marey, subsequently modified and improved by other investigators. It consists essentially of a delicate lever, firmly attached to which is a small button or cushion; the latter, being placed so as to rest exactly on the artery, rises and falls with the expansion and contraction of the vessel and communicates these movements to the lever, which inscribes them on a slip of smoked paper fixed on a plate and carried forwards by clockwork. The more recently constructed instruments of Landois and Sommerbrodt have this advantage over Marey's, that they indicate the precise amount of pressure exerted on the artery. —Only the most important of the vast array of facts brought to light by the use of the sphygmograph, can be mentioned here. It has been ascertained that the *normal pulse* is not monocrotic, but always *dicrotic* (Marey), usually even *tricrotic* (O. Wolff). Thus, whilst the *line of ascent* of *all* arteries, that which corresponds to the expansion or diastole of the arteries, is nearly vertical (varying in height according to the size of the vessel under examination) and *perfectly unbroken*, that which answers to the contraction or systole of the arteries is interrupted by slight secondary *elevations*, of which *two* are more distinctly marked than the others. The first of these owes its existence to the circumstance that the blood-wave, when compressed by the elastic contraction of the artery, is not wholly propagated towards the periphery, a portion of it being sent backwards towards the heart, where it strikes against the already closed aortic valves; the second is produced by the new wave which, generated by this shock, sweeps outwards again in the direction of the peripheral arteries (Buisson, Marey, &c.). These elevations, known as the *recoil-waves*, are the more clearly defined and appear the earlier (that is, the nearer the upper end of the down-stroke of the tracing) the closer to the heart the artery under examination, the less its tension, and the shorter the primary wave (that due to the primary expansion of the artery); the converse of these propositions also holds good (Landois). If both secondary waves occur in the period of contraction of the artery, which is usually the case, the pulse is tricrotic, if one be wanting the pulse is dicrotic. —Besides these recoil-waves several other less marked undulations present themselves in the descending line of the tracing, caused by the oscillations of the arterial wall as it returns to a state of rest after having been expanded by the primary (cardiac systolic) wave, and designated by Landois *elasticity-waves*. They vary in number in different arteries, and are the higher the further removed the vessel in question is from the heart and the greater its tension.

Pathological deviations from the normal form of the pulse tracing are seen sometimes in the ascending, sometimes in the descending line. The up-stroke, which in the healthy pulse is perfectly unbroken and nearly vertical, is occasionally interrupted by a number of small pointed elevations. These are observed when a large volume of blood is projected into the arteries by each systole of the heart, and when the expansion of the vessels is not effected in one movement but in several successive oscillations. Such interruptions of the line of ascent are noticed in the following conditions: in hypertrophy and dilatation of the left ventricle, especially when consequent on nephritis; in those in whom the elasticity of the arteries is diminished,—in old persons, therefore, and in cases of atheromatous degeneration; in aortic stenosis, and in aggravated aortic insufficiency (Landois). The tracing given by the arteries of paralysed parts of the body, where there is vasomotor paralysis combined with a slow circulation, is also of this character.

The pathological modifications of the descending line of the pulse-tracing consist either of an exaggeration or a diminution of the elevations normally present. Should these elevations, particularly the second of those described as recoil-waves, be considerably increased in height, this change becomes *appreciable to the finger* as dicrotism, a sign the special causes of which have already been stated (p. 247). The recoil-waves, on the other hand, are enfeebled when the aortic valves become incompetent, as the regurgitation of blood which takes place into the left ventricle during the cardiac diastole almost always effectually counteracts any tendency to the propagation of such a secondary impulse towards the periphery. In stenosis of the aortic orifice also they are faint and imperfectly developed, on account of the partially filled condition of the arteries; here, however, the slow expansion of the vessels (that is, the slow passage of the current of blood through the contracted orifice) is evidenced by the less upright sloping up-stroke and by the rounding of the apex of the tracing, which normally is distinctly pointed.—The tracings of mitral and other lesions of the heart show little that is characteristic; that given by the small pulse associated with stenosis of the left auriculo-ventricular orifice is certainly throughout less elevated than usual, nevertheless it may be affirmed that on the whole only an approximative diagnosis as to the nature of the heart affection is possible if it be founded solely on the data furnished by sphygmographic examination.

Another instrument, named the "Pulsuhr," which promises to be of great service in the investigation of the properties of the pulse, has recently been introduced by Waldenburg. This is an ingeniously constructed apparatus which enables us to measure with precision, and state definitely in figures, not simply to estimate, as heretofore, the tension, fulness, and volume of the pulse. It is fitted with a mechanism by means of which the position of the plate which rests on the artery and follows its pulsations, may be exactly regulated. The movements of the plate are communicated to a two-armed lever, from the extremity of the longer limb of which passes a thread; the latter winds round an axle and so turns an index, which shows on a dial the distance, magnified about a hundred times, through which the plate is carried; the diameter of the artery, that is, its fulness, is in that way obtained. The same indicator rises and falls with all the movements of the artery; this gives the height of the pulsation, that is, the volume of the pulse, amplified to a similar degree.—To the end of the shorter arm of the lever is attached a spring, the case enclosing which is connected by wheel-work with another index, smaller than that just mentioned, showing on the dial the weight which at any given time presses on the spring. (The construction of the instrument is such that to ascertain this weight the distance travelled by the larger indicator has first to be deducted from that through which the smaller one moves). On turning the screw, the plate having been accurately adjusted on the artery, the spring-case, and with it the lever and the arterial plate, are pushed downwards and both indicators caused to move, the oscillations of the larger of the two corresponding with the height of the pulse-wave. The length of the greatest of these oscillations expresses the *volume of the pulse*. On screwing down the spring-case still further the movements of the indicators become gradually smaller and are finally abolished when the artery is completely flattened and its calibre obliterated: the extent to which the position of the larger index has been altered may now be read on the dial; this gives the diameter of the vessel, that is, its *fulness* at the time of examination; the distance travelled by the smaller index, after subtracting from it the excursion of the larger one, shows the weight which was required to compress the artery, that is, the *tension of the pulse*.

PALPATION OF THE VEINS.

Almost the only veins ever examined by the hand are the jugulars, very rarely the superficial brachial veins; the others yield no diagnostic signs to palpation. The pulsation perceptible in the jugular veins in cases of insufficiency of the tricuspid valves and other affections giving rise to overloading of the right auricle and incompetency of the jugular valves, and the more exceptional forms of pulsation observed in other superficial veins belonging to the domain of the vena cava superior, have already been under discussion; the vibration of the walls of the jugular veins (frémissement or thrill), associated with chlorosis, is reserved for consideration in the section on Auscultation (see "Venous murmurs").

PERCUSSION OF THE HEART.

Percussion of the heart is practised with one of two objects in view,—either the delimitation of those parts of the anterior surface of the heart which, not being covered by lung, are in immediate contact with the chest-wall, or the definition also of those which are situated more deeply, behind portions of the pulmonary tissue. The superficial cardiac dulness may usually be mapped out with a considerable degree of precision; the percussion of the deeper parts, on the other hand, is attended by more difficulty and uncertainty.—It will contribute materially to a clear understanding of the subject if the discussion of details be prefaced by a short description of the position of the heart in relation to the thoracic parietes and the anterior margins of the lungs. The axis of the heart is directed from above downwards, forwards, and to the left, the organ being so placed behind the anterior wall of the chest that two-thirds of its bulk fall to the left of the median line of the sternum and one-third to the right. The highest point of the heart, the uppermost part of the left auricle, is found at the level of a line joining the lower edges of the sternal insertions of the second ribs (right and left); the lowest point of the heart, the apex, lies immediately behind the sixth left costal cartilage, to the inside of the mammillary line. The *left* boundary of the heart, formed by the left border of the organ, corresponds with a line passing from the second left intercostal space downwards to the apex of the heart, describing a curve the convexity of which is turned outwards, and whose greatest distance from the middle line of the sternum amounts to 8—9 ctm. The *right* boundary, which coincides with the right border of the heart, is represented by a line starting from a point in the second right intercostal space close to the sternum, terminating opposite the sternal insertion of the fifth right rib, slightly convex outwardly, and distant 2—3 ctm. from the right edge of the breast-bone. If from the lower end of the last-mentioned line to the apex of the heart another line be drawn, the latter will mark the *lower* boundary of the heart, formed by the wall of the right ventricle.

In the space enclosed within these limits lies the heart, for the most part covered by lung. A certain portion of its surface, however, defined by the anterior margins of the lungs, is not so covered in. As already stated (see p. 108) the lungs are in close apposition anteriorly from the second to the level of the fourth rib, being separated only by the anterior mediastinal fold of the pleuræ. From this point upwards, therefore, the superior segment of the heart,—both auricles and a part of the ventricles,—is completely hidden by pulmonary tissue.

At the level of the fourth rib the lungs diverge, the anterior margin of the left passing outwards through the fourth left intercostal space, turning downwards and a little inwards opposite the fifth costal cartilage, and ending in the lower border behind the cartilage of the sixth rib. The greater part of the right ventricle, and the apex of the left, are thus exposed; the rest of the left ventricle, very little of which indeed appears on the front of the heart, remains concealed.—The anterior border of the right lung turns outwards at a lower point than the left, opposite the sternal insertion of the fifth right costal cartilage; it merges immediately, at the level of the sternal end of the sixth rib, in the lower border of the lung. In following this course it overlaps to a slight extent that part of the right ventricle which lies farthest to the right.

It is thus seen that the segment of the heart between which and the chest-wall no lung-tissue is interposed belongs, with the exception of the apex, exclusively to the right ventricle, and includes little more than the half of that part of the organ. Its extent, also, is not constant; it varies somewhat with the differences frequently observed in the configuration of the lungs. Even in the same individual the movements of respiration are found to affect very considerably the anatomical relations of the heart: during inspiration the edges of the lungs converge and encroach still further on the anterior surface of the right ventricle, and if the inspiration be deep and energetic the entire heart may be so closed in; forcible expiration, on the other hand, causes the margins of the lungs to retire, and a larger part of the heart's surface then comes to the front.

Over the whole of the space described as not occupied by lung, except a narrow portion behind and to the right of the sternum, the percussion-sound is dull; this is termed the *absolute* ir in the pleural sac, when the percussion-sound over the t becomes tympanitic or metallic in quality; or there may luid in the pleura, when the cardiac dulness is indistinguish- from that of the left side generally,—provided the heart be displaced.

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cardiac dulness, or, as it coincides with the more superficial part of the surface of the heart, the *superficial* dulness. It is to be distinguished from the less markedly dull sound given by the rest of the præcordial region, which is designated the *relative* cardiac dulness.

Before proceeding to the percussion of the heart the situation of the apex-beat should first be ascertained, as this indicates at once the inferior limit of the cardiac dulness and thus facilitates its delimitation; the points are then to be determined at which the clear pulmonary percussion-note passes into the dull sound yielded by the heart. The *upper* limit of the *absolute cardiac dulness* begins usually *at the upper border of the fourth left rib;* the *left* coincides with a slightly curved line passing from the superior border of the fourth rib to the apex of the heart, *inside the mammillary line;* the *right* is marked by the *left edge of the sternum from the fourth to the sixth rib.* The lower boundary, which corresponds with the lower border of the right ventricle, is not definable, as the cardiac sound is here continuous with that of the adjacent left lobe of the liver; it may be represented, however, by a line drawn from the sixth chondro-sternal articulation to the spot at which the apex-beat is felt. This space, the shape of which, when traced with black dermographic crayon, is seen to be that of an irregular four-sided figure, is not accurately commensurate with the superficial portion of the heart, as will be obvious on referring to the description already given of the anatomical relations usually subsisting between the heart and the margins of the lungs; to the right of the left border of the sternum lies a part of the right ventricle not overlapped by pulmonary tissue, yet the percussion-note over it (at the middle of the breast-bone, towards its lower end) is not dull, but of the normal pulmonary quality, clear and of low pitch. The cause of this can only be that the sternum, from the readiness with which it enters into vibration, transmits the force of the percussion-stroke to the adjoining lung, so that the slight dulness which the right ventricle really gives is overborne by the clearness of the pulmonary note. This explanation is supported by the observation that on placing the hand on the sternum, and in that way diminishing its power to vibrate, the previously clear sound becomes somewhat muffled.

The extent of the heart's surface not covered-in by lung being, as already noticed, subject to constant variation during the movements of inspiration and expiration, the dimensions of the region of absolute cardiac dulness show similar changes; the limits above set down, therefore, are applicable only to conditions in which the respiration is perfectly superficial, when the differences referred to are so trifling as scarcely to vitiate the results obtained by percussion. It is always necessary, however, to direct the patient to cease breathing when absolute precision in the delimitation of the heart is required, particularly in determining its boundaries superiorly and to the left. Deep inspiration reduces the area of cardiac dulness both in height, as the upper limit sinks downwards, and in breadth, as the left boundary-line moves inwards; only the right border of the region in question remains unchanged.—Whether the patient lies on his back, sits, or stands, is immaterial so far as the accuracy of the results is concerned; but on turning to one or other side the position of the apex-beat is altered and that of the cardiac dulness with it, the latter being diminished to the left and superiorly if the patient be on his right side and enlarged in the same directions if he be on his left. The boundaries of the heart to the right are as little influenced by attitude as by the movements of respiration.

The *intensity of the absolute cardiac dulness* is very inconsiderable, the organ percussed being but small; the stroke must therefore be light, in order to avoid awakening simultaneous vibration in the contiguous portions of the lungs, otherwise the true heart-sound will be lost in the clear pulmonary note. It is necessary to proceed with similar caution in examining the lower part of the præcordial region, particularly in children, as the tympanitic tone derived from the stomach may mask the dull sound belonging to the heart. Simple finger-percussion will be found the method best adapted to the determination of the absolute cardiac dulness.

Those portions of the heart which are overlapped by lung are inaccessible to percussion by the ordinary gentle stroke employed in the manner just described, the sound so elicited being loud and clear; a firm stroke, on the other hand, often demonstrates a decided decrease in the loudness of the note as high as the third rib, indicating that here a thinner layer of lung has been caused to vibrate. Such a sound, therefore, though intrinsically clear in quality, is somewhat muffled, that is, *relatively* dull, as compared with that obtained at other points, behind which only lung is situated. It is chiefly superiorly, towards the upper limit of the præcordial region, that relative dulness is observed on forcible percussion; those other parts of the heart lying behind and to the right of the sternum give no muffling of the sound even in answer to a heavy stroke, and it is only by availing oneself of the sense of resistance communicated to the finger, and at the same time as far as possible eliminating the vibrations of the sternum by pressing on it and in its immediate neighbourhood, that they can be recognised by means of percussion. But even this method fails to demonstrate clearly that portion of the right ventricle which is beyond the right edge of the sternum. To define precisely the *whole* of the anterior surface of the heart is thus impossible; most commonly, therefore, nothing more is attempted than the delimitation of the area which is not sheltered behind pulmonary tissue.—It is obvious that the extent of this superficial cardiac dulness is no absolute guide to the real size of the organ within, as there is no constant relation between the covered and the non-covered parts of the heart, on account of the irregularities so often displayed in the contour of the lungs anteriorly. Nevertheless the dimensions of the region in question usually warrant a general inference as to the presence or absence of enlargement of the heart, as such enlargement is always associated with increase of the area absolutely dull.

In certain circumstances the absolute cardiac dulness may extend beyond the limits above stated, even while the heart remains normal in point of size, as, for example, when the processes of lung which as a rule are found in front of the base of the heart, are atrophied and retracted, permitting the whole of the anterior surface of the heart to come into contact with the chest-wall.

The area of cardiac dulness is, on the contrary, lessened, when the heart, though still of normal size, is shut in to an unusual extent by the lungs. The lungs encroach thus on the heart when their anterior margins are less divergent than is generally the case, or when they are unduly inflated, as in emphysema. The heart may also be pushed backwards and separated from the chest-wall by the accumulation of gas in the pericardium,—a very rare event, when the whole præcordial region renders a clear tympanitic note instead of the normal dull sound; or there may be air in the pleural sac, when the percussion-sound over the heart becomes tympanitic or metallic in quality; or there may be fluid in the pleura, when the cardiac dulness is indistinguishable from that of the left side generally,—provided the heart be not displaced.

The *cardiac dulness is increased* in extent and intensity *in hypertrophy and dilatation of the heart,* this augmentation being most manifest in its *length* in enlargement of the *left* ventricle, and in its *breadth* in enlargement of the *right* ventricle; the dull area further takes a somewhat *conical* form when the *pericardium is filled with fluid.*

In hypertrophy and dilatation of the left ventricle the point at which the cardiac impulse is felt, below and to the outside of its normal situation, offers a ready means of determining approximately the extension of the area of dulness in an outward direction. In such cases also the fixing of the upper limit of the cardiac dulness is exceedingly easy, as the lung, provided it have not contracted adhesions, is pushed aside by the enlarged heart, which then comes closer to, or even touches, the thoracic parietes.

Similarly, in cases of marked hypertrophy and dilatation of the right ventricle the undue propagation of the heart's impulse towards the right at once indicates the nature, and roughly the extent, of the physical change, though usually the non-resonance to percussion, even when the dilatation of the right heart is considerable, is not very apparent beyond the midsternal line.

It often happens that those who have not had much experience in the art of percussion do not consider as dull, or as in any way connected with the heart, the somewhat clearer sound obtained over the sternum in examining from left to right; but on comparing it with the normal pulmonary note, or on percussing still further towards the right till the true lung-sound is educed, the area of dulness associated with enlarged heart is soon seen to be larger than at first suspected.

Slight dilatations of the right heart can be made out only by means of the sense of resistance experienced by the finger in percussing. In cases of marked dilatation of the right ventricle not only does the cardiac dulness far overpass its normal boundaries to the right, but it goes also somewhat beyond them to the left, as is shown by the slight outward displacement of the apex-beat. This is due to the more *horizontal* position which the heart assumes in such circumstances, the necessary result of which is that the apex of the heart is carried further to the left.

Hypertrophy and dilatation of the heart, however, are not in every instance characterised by extension of the præcordial dulness; this sign is absolutely wanting when the overlapping processes of lung are of unusual depth or cover over a larger portion of the surface of the heart. Thus, in vesicular emphysema of the lungs the attendant enlargement of the right heart is frequently unrecognisable by objective examination.

Even with a heart perfectly normal in size, an apparent increase of the area it renders dull to percussion is apt to be produced by various diseases of the adjacent pulmonary texture, such as consolidation (from infiltration, compression, &c.)

It is sufficient merely to mention cases of this kind; to enumerate the signs by which they may be differentiated (certain alterations in the outline of the region of percussion-dulness, &c.,) is perfectly unnecessary, in view of the impossibility of falling into error in the diagnosis. Simple inspection of the præcordial region frequently suffices, as already pointed out, for the positive recognition of hypertrophy of the heart, and the absence of the phenomena formerly described (p. 212 *et seq.*) shows unmistakeably that the increase of the dull area must be due to some other cause.

The area of cardiac dulness is observed to take a peculiar form in cases of pericardial exudation and transudation. If the quantity of fluid be very large, the pericardial sac is fully distended; it is enlarged chiefly in its lower part, while superiorly it gradually diminishes in breadth. The space thus rendered dull is irregularly *triangular* in shape, the *base* of the figure being *downwards*, the *apex* pointing *upwards*. Its size depends on the amount of the exudation; if the effusion be very abundant the base of the triangle may extend from the right parasternal line almost to the left axillary line, while the two remaining sides may be represented by converging lines drawn from the two extremities of the base, meeting in the second intercostal space, and forming there a somewhat obtuse angle. A less considerable amount of exudation is characterised by a proportionately smaller triangle of dulness; in the earlier stages of the affection, also, the non-resonant region has no distinctive outline, dulness being detected both at the base of the heart, where the exudation usually first appears, and in the lower part of the pericardium. The distension of the lower portion of the pericardial sac is recognised by the fact that the dulness extends further to the left than the apex-beat of the heart, a sign which is always a conclusive indication of the existence of pericardial exudation. A very copious effusion completely suppresses the apex-beat; disappearance of the latter, therefore, and a præcordial dulness of the characteristic triangular form, may be accepted as positive proof of the presence of a large pericardial exudation.—And further, as the layer of fluid is so much deeper below (next the diaphragm) than above (towards the roots of the great vessels) the non-resonance is more marked at the lower than in the upper part of the præcordial region; the sound indeed is rendered absolutely dull inferiorly. On causing the patient to lie on his back the fluid at once subsides to the hinder part of the pericardium, diffusing itself equally over its posterior surface, the apex-beat again becomes appreciable, and the percussion-sound somewhat clearer in front, the edges of the lungs being now allowed to expand freely and to fill up the space left vacant by the shifting of the effusion. A similar change in the quality of the sound is noticed on turning to the right or left side: in the former case the sound clears to the left, in the latter it clears to the right.—These statements regarding pericardial exudation apply with still greater force to the more mobile fluid poured out in cases of pericardial *transudation* (hydropericardium).

This variation of the sounds on changing the position of the patient is wanting only when the two opposed surfaces of the pericardium are glued together at several points by inflammatory products, or when the sac is so fully distended that movement of its contents becomes impossible. Such cases, however,—in which the pericardium is rendered so tense by the quantity of fluid it contains as to be incapable of further expansion,—do not seem ever to occur in practice; in one example of enormous hydropericardium which I saw (in which the dulness reached from the left axillary line almost to the right mammillary line, and upwards as far as the third rib) the percussion-sound was found to vary as usual with the position of the patient.

AUSCULTATION OF THE HEART.

The opposite qualities, normal and pathological, of the sounds developed by the action of the heart, are known as *sounds* and *murmurs*.

The Sounds of the Heart.

In normal conditions each contraction of the heart gives rise to *two sounds*, audible over the whole præcordia, and separated from each other by a short pause. The first of these, the systolic sound, is exactly synchronous with the systole of the ventricles, while the second, the diastolic sound, corresponds with the beginning of the diastole of the heart; then follows a pause, the cardiac pause, which lasts till, with the next contraction of the heart, the same phenomena are repeated in the same order.

The sounds are not of equal intensity at all points in the præcordial region: the first sound, that associated with the cardiac systole, is more clearly accentuated at the apex of the heart and over the lower portion of the sternum, than that occurring during the diastole, the rhythm of the sounds at these parts being trochaic; the second is, on the contrary, the more accentuated of the two on the second intercostal space close to the sternum, at both its right and left margins, and the rhythm of the sounds is here iambic. The systolic sounds at the four points just indicated are exactly coincident with each other, as are also the diastolic sounds which succeed them; there are four systolic sounds, therefore, and four diastolic,—eight in all.

These four points on the surface mark, as will afterwards be shown, four centres in or near which the different sounds of the heart originate; *at the apex,* accordingly, will be heard the *sounds produced at the mitral valve, at the lower end of the sternum those connected with the tricuspid valve, in the second left intercostal space, close to the sternum, those proceeding from the pulmonary artery, and in the second right intercostal space, at the edge of the sternum, those generated in the aorta.*

If the heart be displaced downwards, from sinking of the diaphragm, to the extent of one intercostal space for instance, the parts at which its various sounds will be audible will naturally be found at a level corresponding to the abnormal situation of the organ,—one interspace lower; if it be dislocated in any other direction, to the right or left, the apex-beat constitutes a general guide to the points at which the sounds will be heard; where there is no cardiac impulse the points must be sought for at which the sounds are loudest.

The regions described do not correspond absolutely to the anatomical position of the valves of the heart and the large vessels, but it is to them that, as is proved by many physiological and pathological observations, the acoustic phenomena are most freely conducted, and there that they are loudest. This is due chiefly to the anatomical relations of the heart and lungs.

1. Sounds emanating from the *mitral* valve are not looked for directly over the valve (in the second left intercostal space, close to the sternal insertion of the third left costal cartilage), as here the latter lies behind air-containing lung-tissue, a bad conductor of sound; they are auscultated rather at the apex of the heart, which is free of lung and in immediate contact with the chest-wall, and towards which experience shows that sonorous vibrations coming from the mitral valve are transmitted with greatest intensity.

2. In the same way the sounds of the *tricuspid* valve are not to be sought for exactly over their point of origin (behind the sternum, at the level of a line drawn obliquely from the sternal insertion of the third left rib to the fifth right costo-sternal articulation), but somewhat lower down, on the lower portion of the sternum.

3. The *pulmonary sounds* are most clearly conducted to the ear placed precisely over the anatomical site of the pulmonary artery, —in the second left intercostal space, at the sternal insertion of the third left costal cartilage.

4. Sounds developed in the *aorta* are loudest, not just over the orifice of the vessel (in the second left intercostal space), but in the second *right* intercostal space, in the direction of the ascending aorta. As the aorta at its origin completely covers in the root of the pulmonary artery the sounds produced in these vessels are necessarily intermingled, and would be indistinguishable from each other were it not for the fact that those generated at the aortic valves are propagated most energetically in the direction

taken by the current of blood in the ascending aorta, along the course of the vessel towards the second right interspace; at the latter point, therefore, aortic sounds should be auscultated.

It is thus possible to define with sufficient precision the four regions in which the sounds of the heart take their rise, by observing carefully the direction in which these sounds are most perfectly conducted. That this transmission always takes place towards the parts indicated above is proved, provided that the relations of the heart to the lungs be not altered—by the *abnormal* heart-sounds, the cardiac murmurs, to be discussed further on.

It was Læennec who laid the foundation of our present knowledge of the acoustic phenomena developed by the action of the heart; he named the sounds *normal cardiac murmurs*, in contradistinction to the *abnormal cardiac murmurs*. The term *heart-sounds*, as distinguished from *heart-murmurs*, was introduced by Skoda.

Differences in pitch in the heart-sounds are very common. Thus, the second sound at the base is frequently higher than the first, and when the heart is acting with great energy its pitch may often be accurately determined. At the apex the first sound is most usually deeper and duller than at the lower part of the sternum, where it is sometimes exceedingly loud and clear; this is obviously to be explained by the facility with which sound is conducted through the solid tissue of the sternum. The sounds also present great variety in timbre, being sometimes clear and ringing, at other times dull and without proper musical character; even in the same person this timbre may change rapidly with the varying force of the heart's contractions.

The *rhythm* of the sounds at the apex and over the lower portion of the sternum (that is of the sounds of the left and right ventricles) is not invariably trochaic, nor is it always iambic at the roots of the great vessels; often it is the reverse of this, trochaic over the vessels and iambic at the apex, or in both situations iambic, or in both trochaic. These differences depend on the occasional *predominance of the first sound*, when the rhythm is at all points trochaic, or *of the second sound*, when the rhythm becomes iambic. These modifications have no particular significance.

If the action of the heart become irregular from any cause the rhythm of its sounds is also lost; in the severer forms of irregularity, indeed, it is a matter of some difficulty to distinguish between the systolic and diastolic sounds.

PHYSICAL CAUSE OF THE HEART'S SOUNDS.

The *first* sound arises from the *tension of the auriculo-ventricular valves*, due to the shock of the mass of blood thrown against them during the cardiac systole; the *second* sound is caused by the *tension of the semilunar valves of the aorta and pulmonary artery* at the instant that the blood projected into the vessels is driven backward by the elastic recoil of the arterial walls.

During the period of rest the auriculo-ventricular valves hang loosely down into the ventricular cavities, but are floated upwards when the latter are filled with blood and belly out in the direction of the auricles, shutting off the lower from the upper chambers of the heart; the short auricular systole, which precedes by an instant that of the ventricles, renders them somewhat tense, but to such a slight degree as to give rise to no audible sound. Sound is produced only by the more marked tension which results from the systole of the ventricles, and which is caused partly by the pressure of the blood against the valves, and partly by the contraction of the papillary muscles connected with the valves by the chordæ tendineæ.

The theory above stated, first propounded by Rouanet (1832), is supported by many physical analogies and pathological observations. Every elastic membrane (like a cord of cat-gut, for instance) emits a sound on being suddenly brought from a state of relaxation into one of extreme tension; the sudden tension of such membranes, therefore, as the cardiac valves, may naturally be supposed to be attended by similar phenomena. Experimentally, also, a feeble, dull tone may actually be obtained from a tense mitral valve by directing against it a forcible stream of water either from the apex of the heart (O. Bayer) or through the aorta, the semilunar valves of the latter being previously removed (Landois).

The mitral and tricuspid valves must also give out sound, each independently of the other, but both at the same moment, both ventricles contracting simultaneously.

Further proof of the accuracy of Rouanet's theory is furnished by certain pathological conditions. Thus, if the mitral valve lose its smoothness of surface and delicacy of structure from pathological deposits of any kind, from retraction or inflammatory adhesion of the tips of the valvular segments, &c., in such a way that it becomes totally or partially incapable of being put thoroughly on the stretch, the systolic sound at the apex of the heart undergoes change, or disappears completely and is replaced by a murmur. Over the lower third of the sternum, however, the first sound remains perfectly pure and clear so long as the tricuspid valve retains its smoothness,—which it usually does.

On the other hand, in those rare cases in which the tricuspid valve has been deprived of its elasticity, the systolic sound over the lower portion of the sternum is superseded by a murmur, while at the apex it is still distinctly heard provided that the mitral valve be intact.—

But tension of the auriculo-ventricular valves, though the principal, is not the only cause of the first sound of the heart; this depends to a certain extent also on the *muscular contraction* of the ventricles. Every large muscle, when contracting vigorously, especially under electric stimulation, generates sound (Wollaston), usually of a quality so distinctly musical that its pitch may be determined with precision (Helmholtz). This takes place not only in all striped muscles, but also in the heart. The most weighty evidence in favour of this view, however, is presented in the experimental fact that the heart, removed from the body and emptied of blood, emits a distinct sound at each contraction (Ludwig and Dogiel), although under these conditions the tension of the auriculo-ventricular valves is reduced to a minimum.

And further, in the domain of the pathology of the heart many phenomena are observed which tend to support the doctrine that muscular contraction plays a part in the production of the first sound. Thus the systolic sound is found not to be invariably abolished by the presence of even very marked and advanced changes in the mitral valve; and, on the other hand, it may become indistinct and feeble, even though the mitral valve preserve its normal smoothness and general structure, if the *muscular substance* of the heart be affected, as in myocarditis, fatty degeneration, and other diseases which diminish the contractile power of the organ.—There are also other circumstances which lend countenance to the theory that the action of the cardiac muscle is concerned in the generation of the first sound,—the facts that in certain conditions the duration of this sound is longer than can be satisfactorily accounted for by the transient tension of the auriculo-ventricular valves, and that a simple state of tension of the mitral valve, produced artificially (by water-pressure) in the heart extracted from the body, gives rise to a sound having no resemblance to that heard in the living subject. Nevertheless, the part taken by muscular contraction in the development of the systolic sound of the heart is but a very subordinate one: it must not be inferred,—solely on the experimental ground that the heart even when empty gives a sound in some respects similar to the normal first sound in the living subject,—that muscular contraction is the essential factor in the causation of the sounds and the tension of the valves but a secondary element, as such a conclusion is plainly at variance with the fundamental phenomena presented in cases of cardiac valvular lesion.

It is now no longer necessary to discuss or refute in detail the numerous theories that formerly prevailed regarding the cause of the first sound of the heart; the only question that calls for particular notice is concerning the greater or less share taken by muscular contraction in the production of this sound. Till comparatively lately the sudden tension of the auriculo-ventricular valves was almost universally accepted as the sole cause of the systolic sound, the arguments advanced by certain of the earlier authors (Lænnec, Williams, Hope, &c.,) to show that the cardiac sounds are simply muscular, meeting with but partial acceptance; recently, however, Ludwig and Dogiel, basing their views on experimental research, have again ascribed to the cardiac muscle the principal rôle.

I have repeated Ludwig and Dogiel's experiments, and in doing so pursued the following method: the circulation was controlled by opening the thorax (artificial respiration being carried on) and enclosing the great vessels entering and leaving the heart in a ligature, in such a way that the current of blood could be arrested or restored at pleasure. Like Ludwig and Dogiel I found that the empty heart, at each contraction, certainly emits a sound which, as might have been expected from the feebleness of the heart's action in such circumstances, is of far less intensity than the normal heart-sound; I have to state positively, however, that *this sound differs essentially in character from that of the normal heart, it is duller, is toneless* (*without timbre*), and wants also the flapping quality which distinguishes the ordinary first sound.—Another source of error in these investigations remains to be pointed out, namely, that in the empty heart also the musculæ papillares continue to contract, and that therefore a certain, though probably slight amount of tension of the auriculo-ventricular valves is brought about. There is thus the possibility to be borne in mind, that this valvular tension may be sufficient to give rise to a sound which, though feeble, goes to diminish still further the relative importance of muscular contraction in the causation of the first sound.—The only really satisfactory proof that the contraction of the cardiac muscle has any claim to be regarded as an influential factor in the origination of the first sound would be the production of evidence to the effect that the sound is heard even after total *destruction* of the auriculo-ventricular valves; it seems impossible, however, to perform such an operation on the living animal without at the same time setting up such grave disturbance as would inevitably vitiate the result of the observation.

O. Bayer adopts the theory that the sound is muscular, adducing certain clinical and pathological facts in support of that view. Thus, he states that very frequently in severe acute and chronic diseases he has noted various changes in the first sound, especially enfeeblement and diminution in its distinctness, while, as was demonstrated by *post mortem* examination, not the slightest trace of valvular affection existed; there was invariably present, on the other hand, a more or less considerable degree of alteration of the muscular structure of the heart, (particularly fatty degeneration of the primitive bundles and albuminous infiltration) occasionally plainly visible to the naked eye, at other times to be detected only with the aid of the microscope.

In all these cases a much less forced conclusion appears to me to be indicated, namely, that the modifications of the cardiac sounds referred to are due rather to *non-uniformity in the vibrations of the valves* (see p. 287). There are also on record various instances in which, notwithstanding the fact that the cardiac muscular tissue was the seat of extensive degeneration, and thus quite incapable of originating any sound, the sounds of the heart were normal during life, and could be caused only by tension of the unaffected auriculo-ventricular valves (Bamberger).

It is further often asserted, in favour of the muscular theory, that mitral lesions grave enough to render the valve absolutely *insufficient* do not always cause the first sound of the heart to disappear; in such conditions it is only masked by the systolic murmur, through which it can always still be heard on careful auscultation.—I find, as the result of my own personal experience in nearly 200 cases of mitral insufficiency, that in these circumstances the systolic sound very rarely continues to be audible; and I would also submit that even when it does persist, notwithstanding the existence of a systolic murmur, we have in this fact no absolute proof of its muscular origin. In the first place, it is never so loud as that heard over the right ventricle, (the tricuspid valve being intact); and further, the mitral valve may, if there still remain certain portions of its tissue not invaded by the degenerative process, preserve its power to vibrate at those parts, and may in this way be enabled to emit a sound; or a systolic sound may be conducted from the right ventricle to the apex of the heart, a consideration which would explain the persistence of the sound along with a systolic murmur even in those cases in which the mitral valve is degenerated in its whole extent.

There still remains to be mentioned another phenomenon, observed in the domain of cardiac pathology, which admits of no possible explanation on the supposition that the first sound is muscular, and which is compatible only with the valvular theory,—the *absence* of the systolic sound at the apex when the aortic valves are to a high degree incompetent. Were the first sound due to muscular contraction it should still be heard in such cases, or should even be louder than usual, inasmuch as the insufficiency of the aortic valves leads to hypertrophy of the left ventricle. Now in hypertrophy of the left ventricle from any other cause, as from disease of the kidneys or sclerosis of the aortic system, the first sound is clearly marked, frequently intensified; it is unquestionable that such enlargement of the heart not only does not render its sounds weaker, but actually renders them *stronger*,—though some have indeed held, on the contrary, that a hypertrophied ventricle is less capable of producing a sound than one of strictly normal development. The absence of the first sound in aortic insufficiency, however, is, as Traube has shown, readily explained by the valvular theory of the origin of the sounds. Thus, during the diastole the auriculo-ventricular valve, the mitral for example, is in a state of relaxation; at the end of the diastole it is put slightly on the stretch by the contraction of the left auricle, as has been already stated on p. 263. This slight degree of presystolic tension (*initial tension*) gives no sound; an audible sound is produced only by the firmer tension which results from the systole of the ventricle (*final tension*). The greater the difference between the initial and the final tension of the auriculo-ventricular valves the louder will be the sound, just as in any other membrane which, previously slack, is *suddenly* and violently tightened. If, on the other hand, the difference between initial and final tension become less, the vibrations of the valve become less ample and the sound consequently more feeble; and if the difference be diminished still further, absolutely no sound is heard. Diminution of the difference between the initial and final tension of the mitral valves of precisely the nature described, is observed in cases of aortic insufficiency: the initial tension increases, as at the end of the diastole of the left ventricle the blood rushing back from the aorta presses on the valvular segments, while the final tension is lessened, as in this affection the aorta, into which the left ventricle projects its contents, soon loses much of its normal elasticity and tension.

Some have endeavoured to explain the absence of the first sound in aortic insufficiency as owing to fatty degeneration of the muscular substance of the heart; but this view is negatived by the fact that it is not only in the later stages of the valvular affection, when the contractile power of the heart begins to fail, but also in its earlier stages, when the hypertrophy of the ventricle is *fully* compensatory for the valvular defect and the action of the heart regular and powerful, that the systolic sound is wanting. Moreover, in cases of genuine fatty heart, in which the degeneration of the primitive muscular bundles is unquestionably much more pronounced than when the structural alteration is consequent on aortic lesion, the first sound is generally clear, though possibly feeble.—Other less weighty objections to the valvular theory may be passed over without further discussion.—Although the whole question in dispute cannot yet be said to be definitely settled, the conclusion to which the present state of our knowledge seems to point is that *the first sound is essentially of valvular origin and only to a slight extent muscular.*

CAUSE OF THE SECOND SOUND OF THE HEART.

That the second sound of the heart is produced, not within the chambers of the organ, but at the semilunar valves of the aorta and pulmonary artery by the recoil of the column of blood against these structures, and that it is only conducted towards the cavities of the heart, is placed beyond all doubt by numerous observations, experimental as well as pathological.

The second sound disappears totally on cutting off the flow of blood to the heart in living animals. This may be effected, (artificial respiration being kept up) by passing a ligature round the venæ cavæ close to the point at which they enter the right auricle; on lifting and thus tightening the cord circulation is stopped, on slackening the cord it goes on as before. Raising the ligature abolishes the sound completely, lowering the ligature restores it at once; but the first sound is found to persist, notwithstanding the arrest of the circulation, as the heart continues to contract as usual and the auriculo-ventricular valves are regularly thrown into a state of tension. Over the heart, also, removed from the body and still pulsating, only the first sound is heard, not the second; after the heart has ceased to contract, however, a second sound may be produced at the aortic valves by closing them abruptly by injecting a stream of water into the vessel with sufficient force. The greater the pressure exercised by this body of water the louder the sound obtained.

The ordinary physiological and pathological phenomena which pass under our notice every day are strongly confirmatory of the above conclusions. Thus, it is observed that the second sound is much feebler over the ventricles than opposite the arterial orifices, and it is only at the latter situation that it possesses its distinctive flapping character. While auscultating, if the stethoscope be slowly moved along the surface from the region of the apex towards that corresponding with the orifices of the great vessels the gradual intensification of the second sound may often be clearly traced.

But by far the strongest evidence that the second sound of the heart is generated at the semilunar valves is furnished by the following pathological observations. When, from hypertrophy of the *left* ventricle, the blood is thrown into the aorta with unusual force the second sound becomes abnormally loud, in consequence of the more energetic recoil of the mass of blood against the aortic valves. When, on the other hand, the quantity of blood which enters the aortic system on each contraction of the heart is small, as in mitral insufficiency, and more especially in mitral contraction, the diastolic recoil of the blood against the aortic valves is less forcible and the second sound becomes much fainter, or occasionally even quite inaudible, over the left ventricle. This occurs also when the contractile power of the heart is from any cause lowered, as in cases of fatty degeneration, the general failure of strength which precedes death, and in the asphyxial stage of cholera. (In the last-mentioned affection there is sometimes not the slightest indication of a second sound at the apex, while it is still perceptible, though faintly, at the great arterial orifices.) Further, when the aortic valves are so altered in structure as to be no longer competent to perform their office efficiently, when therefore there is no second sound but a distinct murmur at the root of the aorta, the diastolic sound over the left ventricle is also wanting, nothing whatever being heard after the systolic sound, or only the feeble diastolic murmur conducted from the aortic valves. And finally, in cases of reduplication of the second aortic sound, the second sound over the left ventricle is also double, but weaker than over the aorta.

It can be shown equally clearly that the second sound heard over the *right* ventricle is transmitted from the semilunar valves at the origin of the pulmonary artery. If, as the result of hypertrophy of the right ventricle, the blood be driven into this vessel with greater force than usual it is thrown back with proportionately greater force against the semilunar valves during the diastolic period, and the second pulmonary sound is consequently intensified; over the right ventricle also the same sound is then considerably louder. If, on the other hand, the pulmonary valves become insufficient from degeneration,—a somewhat rare occurrence,—no second sound, but a diastolic murmur, is produced, and over the right ventricle also there is no second sound, but the feeble diastolic murmur conducted from the pulmonary valves. And finally, when the second pulmonary sound is double at its point of origin it has the same character over the right ventricle.

Cause of the First Arterial Sound.

The first arterial sound, which is heard in the second right and second left intercostal spaces, close to the sternal articulations of the third pair of ribs, and which is synchronous with the first ventricular sound, is to some extent to be regarded simply as the ventricular sound propagated towards the periphery; it *takes its origin chiefly, however, at the orifices of the aorta and pulmonary artery, and is due to the tension and sudden expansion of the arterial walls which take place when the blood is discharged into the vessels by the contraction of the heart.* Many pathological observations go to prove this. Thus, if the coats of the first part of the aorta undergo such changes (as from atheromatous degeneration) as deprive them of their property of uniform expansibility and of their power of entering into vibration, the arterial sound under consideration is not developed; instead of it is heard, at the aortic orifice, a systolic murmur, while the first ventricular sound remains unaffected. Similarly, the first sound at the pulmonary orifice disappears, and is replaced by a murmur, when the width of the opening is diminished or the inner surface of the vessel roughened by disease.—The first sound at the arterial orifices also continues audible when over the ventricles, generally over the left, the first sound is abolished or is represented by a systolic murmur.

But it is not merely in the central portions of the vessels springing from the heart that this systolic sound, arising from distension of their walls by the advancing blood-wave, is observed; it occurs also in their larger subdivisions, in the carotid and subclavian, and even, under certain pathological conditions, in still smaller arteries, at a greater distance from the heart. These sounds will be treated of when discussing the auscultation of the arteries.

It will thus be seen that *of the eight sounds* heard at the above-described four points in the præcordial region *to six only is a proper point of origination assignable: at each of the auriculo-ventricular valves one (systolic) sound is produced; in the aorta and pulmonary artery arise two sounds, one systolic and one diastolic;* the second ventricular sound is merely the transmitted second arterial sound.

These views regarding the parts at which the heart-sounds are developed, first announced by Rouanet and accepted (with some modification only with respect to the first arterial sound) by Skoda, have been almost universally adopted.

The diagnostic interpretation of the presence of the normal cardiac sounds is simply this,—that all the valves are faithfully executing their normal functions, and that therefore there is no obstacle to circulation within the heart itself. But the satisfactory performance of their functions does not necessarily imply the absolute anatomical integrity of the valves themselves; slight changes may nevertheless exist, and these are not unfrequently found on examining the bodies of individuals in whom the cardiac sounds during life were perfectly clear.

PHYSIOLOGICAL AND PATHOLOGICAL VARIATIONS IN THE CHARACTER OF THE HEART-SOUNDS.

The sounds of the heart naturally present so much diversity of character, and pass from the domain of health to that of disease by stages so insensible, that it is frequently only by calling to our aid such other signs as are furnished by an exhaustive examination of the organs of circulation, that it can be determined whether or not these changes in the sounds are really due to anatomical lesion of the heart itself. These alterations affect the *intensity*, the *purity*, and the *timbre* of the sounds; *reduplication* or *division* of the sounds comes under the same category.

THE INTENSITY OF THE HEART-SOUNDS

is very different in different individuals, even in the total absence of any excitement of the heart's action. The sounds are somewhat louder in the erect than in the recumbent posture, and are still more accentuated when decubitus is on the left side; they also acquire greater intensity during expiration and in the respiratory pause, as in inspiration the margins of the lungs creep forward and cover the heart and so prevent the free conduction of its sounds to the surface. The strength of the cardiac sounds is also considerably modified by the thickness of the thoracic parietes; other things being equal the sounds are therefore louder in children and in those who are emaciated than in persons having large muscles and an abundant covering of fat over the chest. They are further intensified by everything which increases the force of the heart's contractions,—mental excitement, physical exercise, the febrile state; in these circumstances *all* the sounds, both those connected with the ventricles and those emanating from the vessels, are alike influenced.

Intensification of only one of the diastolic arterial sounds, however, caused by hypertrophy of the heart, is very frequently observed, either in the region of the aorta or of the pulmonary artery. To determine whether there is actually any exaggeration of either sound it is only necessary carefully to compare the one with the other. Normally the second aortic sound is a very little louder than that of the pulmonary artery. Under pathological conditions the second sound at the two great arterial orifices varies in strength within very wide limits; the reinforcement of the sound may even be so considerable that the diastolic recoil of the blood against the semilunar valves, which gives rise to the sound itself, may be perceptible to the hand or to the eye as a distinct pulsation in the superficial parts corresponding to the situation of these vessels (see p. 238).

Hypertrophy of the left ventricle, from whatever cause arising, (excepting only that form which results from aortic insufficiency, as in such circumstances no sound is produced, but a diastolic murmur), *intensifies the second aortic sound.*

An *intensification of the second pulmonary sound* is the necessary consequence of *hypertrophy of the right ventricle,* to whatever cause the latter may be due, with the exception of those exceedingly rare cases in which the enlargement is connected with insufficiency of the pulmonary valves, when the diastolic sound is suppressed and replaced by a diastolic murmur. The most notable increase in the loudness of this sound takes place in hypertrophy of the right side of the heart from mitral insufficiency or stenosis of the mitral orifice, or from a combination of both lesions; it is as a rule more marked in the young than later in life.

The degree to which the second pulmonary or aortic sound is exaggerated in intensity is generally directly proportionate to the enlargement of the corresponding ventricle.—When, from fatty degeneration of the muscular substance of the heart, the contractile power of the hypertrophied ventricle begins to fail and to be inadequate to fully compensate for valvular defects, the strength of the second arterial sound also diminishes.

It was Skoda who first drew attention to exaggeration of the second pulmonary arterial sound as a positive and unerring indication of mitral valvular lesion (in the stage of compensation). The same phenomenon is observed, though less strikingly developed, independently of defect of the mitral valve, in every variety of congestion of the pulmonary circulation, as this commonly leads to hypertrophy of the right heart (*e.g.*, bronchial catarrh of long duration accompanied by vesicular emphysema). It is in mitral affections, however, that it is found in its most marked form, as in such cases the consecutive hypertrophy of the right heart reaches its maximum.

The increased loudness of the second arterial sound is the more distinctly audible the fewer and the less important the obstacles to the transmission of the sounds to the walls of the chest; it is thus, other things being equal, most declared when the anterior border of the lung recedes and exposes entirely the base of the heart, as so often happens when the heart is very considerably hypertrophied in young persons, the base of the organ being then in immediate contact with the front of the chest. If, on the contrary, the heart be completely closed in by pulmonary tissue, whether from adhesion of the anterior margin of the lung in such a manner that it cannot retreat or from emphysematous distension, the second pulmonary arterial sound may be of merely its normal strength notwithstanding the presence of very decided hypertrophy of the right side of the heart.—It is further a common experience to find this sound of greater intensity when the upper lobe of the left lung is consolidated and excavated, and that without any enlargement of the right heart; the first pulmonary sound then also appears to be somewhat louder than the first aortic sound. In such cases, however, the pulmonary sounds are not in reality louder than those originating at the aortic valves, they are merely carried to the surface with greater precision through the solid lung parenchyma. Moreover, in those instances in which the consolidated portion of lung shrinks in volume a large part of the *base of the heart* lies immediately behind the thoracic wall.

Exaggeration of the second arterial sound is of much less frequent occurrence in the aorta than in the pulmonary artery, as simple and uncomplicated hypertrophy is less common on the left side than on the right, and as, further, it is precisely in the affection which most often causes enlargement of the right ventricle,—aortic insufficiency,—that the diastolic sound is masked by a diastolic murmur. Almost the only disease of the heart, therefore, which results directly in hypertrophy of the left ventricle and intensification of the second aortic sound is simple contraction of the aortic orifice unattended by lesion of the aortic valves, —a condition which is on the whole seldom met with. The most exquisite examples of exaggeration of the second aortic sound are observed in hypertrophy of the left heart from atrophy of the kidneys or arterial sclerosis, as in these cases the valves and the orifice of the aorta remain perfectly normal.

ENFEEBLEMENT OF THE SOUNDS OF THE HEART.

This is frequently noticed, within physiological limits, in perfectly healthy persons and in those whose chests are protected by a thick covering of fat. Pathologically it proceeds from one of three causes: from the mere feebleness of the heart's action, —in cases of general debility, therefore, in convalescence from severe acute diseases, and in fatty degeneration of the cardiac muscular substance; from the presence of some obstacle to the clear transmission of the heart-sounds to the chest-wall, as when the margin of an emphysematous lung comes in front of the heart, and when the latter is separated from the thoracic parietes by pleuritic or pericardial effusion; or from masking of the sounds by loud râles in the adjoining parts of the lungs.

All the cardiac sounds are weakened in the conditions just named. But there are also cases of cardiac valvular lesion in which the second arterial sound alone is effected; the sign, however, is of no great diagnostic importance. Thus, the partially-filled state of the aortic system which accompanies extensive and advanced mitral disease (insufficiency and stenosis) leads to enfeeblement of the second aortic sound; and in the rare cases in which the tricuspid valve becomes incompetent the diminution in the quantity of blood in circulation in the pulmonary system lessens the intensity of the second pulmonary arterial sound.

HEART-SOUNDS ALTERED AS REGARDS THEIR PURITY.

The sounds of the heart, particularly the systolic ventricular sounds, are frequently neither so accentuated nor so markedly flapping in character as those heard in health; they become impure, and in extreme instances lose more or less completely the qualities of a sound proper, so much so that one is sometimes in doubt whether to regard them as sounds or murmurs. Sounds of this doubtful kind often merge into unmistakeable murmurs when the heart is excited to more powerful action.

The slighter degrees of impurity of the sounds may have their origin in comparatively unimportant changes in the auriculo-ventricular and arterial valves; thus a merely trifling diminution in the structural delicacy of the valves, a slight thickening of their substance, perhaps also the want of absolute uniformity of tension and vibration in the various valvular segments, and other similar but not yet thoroughly known conditions, may be sufficient to produce such a result.

Impurity of the Sounds, when not associated with other cardiac anomalies, particularly with changes in the cardiac impulse or in the volume of the heart, has not usually any special diagnostic value.

Heart-Sounds of Ringing, Metallic Quality.

These are observed when air gains access to, and accumulates in the pericardium (*pneumopericardium*), in left pneumothorax, and when there are large air-filled cavities in those parts of the lungs which are in immediate proximity to the heart. In all these cases a metallic timbre is added to the heart-sounds, by consonance in the air-spaces through which they are conducted (compare the metallic respiratory murmur and râles, pp. 139, 156, *et seq.*).

The heart-sounds acquire a ringing metallic timbre of the most exquisite quality in pneumopericardium. The air may enter the pericardial sac from without through wounds, or from within through pathological communications established between parts containing air and the sac, as from perforation of the œsophagus or stomach, or the bursting of a pyopneumothorax or pulmonary cavity into the pericardium, or, as in one case I saw in Traube's clinique, the gas may be given off by a pericardial exudation.

In left pneumothorax the sounds have not invariably the metallic character; it is wanting particularly when the heart is much displaced towards the right side. The amount and tension of the air in the pneumothoracic cavity modify this sign in many ways. It is only under specially favourable circumstances also that pulmonary cavities situated near the heart communicate to the sounds a metallic timbre.

Whilst in the pathological conditions just mentioned *all the heart-sounds* have the ringing quality, in cases of *atheromatous* degeneration limited to the initial portion of the aorta the second aortic sound alone is frequently of this character, so long as the valvular segments at the root of the vessel remain perfectly intact.

Reduplication or Division of the Heart-Sounds.

The systolic and diastolic sounds may each be broken up into two distinct parts; if these be separated from each other by a short [illegible] ey may be spoken of as *reduplicated* cardiac sounds, or if the one passes into the other without any appreciable interval, as *divided* sounds. Reduplication and division, however, are terms which are generally used synonymously, as both forms commonly owe their existence to the same causes, and are apt to pass insensibly into each other even in one and the same patient. For instance, the pause which comes between the two elements of the sound disappears when the heart's action is accelerated, and the properly double sound becomes a divided sound. The closer the two parts approach to each other the less clear and pure is the quality of the sound. The rhythm of the heart-sounds when the first is reduplicated is anapæstic, when the second sound is so affected, dactylic.—In some rare cases the sound falls into three very short portions.—Division of the first ventricular sound is relatively most frequent, the next most common phenomenon of this kind being doubling of the second arterial sound.

Reduplication of the *first* ventricular sound occurs in perfectly healthy persons, but is then never permanent,—it comes and goes, disappearing entirely as the force and rate of the heart's action are increased. It is noticed temporarily also in diseases of the heart, but cannot be said to be characteristic of any particular affection. It is connected sometimes with mitral and sometimes with tricuspid disorder, and most probably originates in *non-synchronous tension* of the individual segments of the auriculo-ventricular valves. This explanation does not necessarily involve the assumption of the non-simultaneous contraction of the ventricles, though such an occurrence is not impossible in certain cases, marked by a double cardiac impulse (see p. 208); the irregularity in tension of the valvular segments may quite justifiably be ascribed to the absence of perfect uniformity in the contraction of the papillary muscles. When the heart begins to act more *powerfully* these muscles contract regularly and energetically, the tension of the valves with which they are connected becomes uniform, reduplication of the sounds vanishes, and the two sounds become *one*.

Two other explanations, much less probable than the one here adopted, have been advanced to account for doubling of the first sound.

1. It is explained by some on the supposition that the tension of the arterial walls produced by the entrance of the systolic blood-wave is later in point of time than the tension of the auriculo-ventricular valve. But in the first place, the interval which separates these two phenomena

is so exceedingly short that the formation in this way of two systolic sounds, often marked off from each other by a very appreciable pause, is scarcely possible; and in the second place, even in health there is always such an interval, though it is not directly measureable, between the systole of the heart and the expansion of the aorta and pulmonary artery, whilst the occurrence of a double first sound is far from common and is only periodic in those in whom it does appear.

2. Reduplication of the first sound has further been said to be due to the non-simultaneous contraction of the two ventricles, a similar irregularity being thus established in the tension of the mitral and tricuspid valves. But in these circumstances the second arterial sound should also always be doubled, for if the ventricles do not contract in concert arterial expansion, and consequently the diastolic closure of the semilunar valves, should likewise be non-simultaneous. Moreover, both portions of the divided systolic sound are exactly alike in intensity and timbre, which would not be the case if they were propagated from different ventricles. And lastly, this theory fails entirely to explain those rare cases in which the first sound is split up into three parts (*trommelschlag*, drum-beat).

In view of the fact that reduplication of the systolic sound of the heart is not very rare even whilst the organ itself is healthy, no very decided diagnostic signification can be attached to the sign. I have on several occasions observed that it could be artificially produced by pressing firmly over the apex of the heart in those in whom there was already some impurity of the first sound. Potain has also, in cases of hypertrophy of the heart consequent on granular atrophy of the kidneys, met with a variety of double first-sound (*bruit de galop*) in which besides the two normal sounds a third was heard, coming immediately before the first sound and separated from it by a short pause; this additional sound was therefore presystolic. It is quite conceivable that this presystolic sound, which I too have noticed in a number of instances of hypertrophy of the heart, is caused by the contraction of the hypertrophied auricles. The auriculo-ventricular valves are to a certain extent rendered tense even at the end of the diastole, that is, in the presystole, but this tension is normally so feeble that no sound results; but if the walls of one of the auricles undergo hypertrophy as the consequence of some valvular lesion, the corresponding auriculo-ventricular valve is put more sharply and thoroughly on the stretch by the contraction of the auricle and in this way the conditions necessary to the production of a presystolic sound are realised. The systole of the heart is then followed by another sound, dependent on the much fuller tension of the valve, and considerably louder and clearer in tone than the presystolic sound in question. — I have frequently heard doubling of the first sound associated with a systolic murmur; occasionally also I have observed this sign at once develope into a marked systolic murmur when the heart's action was increased in force.

Reduplication or division of the *second arterial sound*, audible over the aorta and pulmonary artery, occurs sometimes physiologically, but more generally as a pathological condition. Its cause is *non-simultaneous closure of the pulmonary and aortic valves.* The two diastolic arterial sounds therefore do not coincide with each other, and one of the two heard over either of the arteries is thus invariably conducted from the other vessel. It is, nevertheless, not unreasonable to suppose that certain anatomical changes in the semilunar valves might cause the tension of these structures to take place in two distinct movements, and thus give rise to division of the second sound; this assumption would explain those cases in which the reduplication is confined to the sound proceeding from only *one* of the arterial orifices, while that arising at the other is either simple and undivided or very much enfeebled in its second element.

Reduplication of the second sound occurs most often, and is most fully developed, in *stenosis of the mitral orifice.* In my experience it is heard in almost a third of all such cases. It is most clearly defined over the lower portion of the sternum and near the apex of the heart, and is less pronounced in the region of the arterial orifices; it is, further, generally appreciable only when the heart's action is slow and tranquil, while if the heart be excited to quicker and more vigorous contraction the two sounds usually merge into a diastolic murmur: in some cases, however, they persist, notwithstanding the appearance of this murmur. The double diastolic sound associated with mitral contraction remains tolerably constant, and always returns even after a temporary disappearance.

Reduplication of the second sound of the heart in mitral stenosis is difficult to account for satisfactorily. Geigel ascribed it to non-coincidence of the closure of the arterial valves, the necessary consequence of the unusual difference in the quantity of blood contained by the aorta as compared with that in the pulmonary artery in cases of this affection; the aorta receives a relatively small quantity of blood on each contraction of the heart, is therefore but slightly distended, and accordingly contracts sooner than the pulmonary artery and its valves are sooner closed than those at the entrance to that vessel; the latter also is constantly overloaded with blood discharged by the hypertrophied right ventricle and is thus unduly distended, and its walls are eventually to a considerable extent deprived of their elasticity.

There are several circumstances, however, which seem to tell decidedly against this view: the broken diastolic sound is (so far as I have observed) certainly not loudest over the large vessels, but at the lower part of the sternum and near the apex of the heart, and is, further, absent in the more marked cases of mitral contraction, precisely the cases in which the conditions most favourable to the postponement of the closure of the pulmonary valve are present in the highest degree; division of the diastolic sound, also, is never met with in mitral *insufficiency*, notwithstanding the fact that it is followed by the same consecutive changes (hypertrophy and dilatation of the right ventricle, engorgement of the pulmonary circulation) as stenosis. It appears rather that the reduplication of the second sound originates at the narrowed orifice itself, as it so often vanishes completely on increasing the force of the heart's action, and gives place to a loud diastolic murmur; there is therefore nothing forced in the inference that the two elements of the phenomenon in question, which form a sound which is always more or less muffled or impure, are in reality the component parts of a murmur.—It has also been conjectured, on the other hand, that the first part of the divided sound is simply the diastolic pulmonary sound, and that the second is produced, towards the end of the diastole, by the contraction of the hypertrophied left auricle,—a theory which would yield a plausible explanation of those cases in which the reduplication remains even when the diastolic murmur is developed.

A doubled second sound is heard also when the *pericardium becomes adherent to the heart* (Friedreich); here the posterior wall of the chest, being first dragged inwards towards the vertebral column during the cardiac systole, springs back sharply to its original position during the diastole, the parietal vibrations so generated constituting a dull muffled sound following closely on the second ventricular sound.

CARDIAC MURMURS.

The murmurs heard over the heart, in pathological conditions, arise either *within* the heart or the initial portion of the great vessels, or *outside* the heart, on its outer surface; the first are named *endocardial*, the second *pericardial*, murmurs.

ENDOCARDIAL MURMURS.

These are produced,

1. By those anatomical changes in the valves or arterial coats which give rise to incompetence of a valve, to contraction of a valvular orifice, or to dilatation of the roots of the great vessels, lesions which all cause some obstruction of the circulation; occasionally, however, they are produced also by anatomical changes which offer no impediment to the passage of the blood, such as the presence of deposit on the endocardial lining of the ventricles, &c.

2. By non-uniformity in the tension of valves and arterial walls, the anatomical structure of the heart being perfectly normal.

Murmurs dependent on the existence of actual obstruction of the circulation are termed *organic* murmurs, those occurring independently of such obstacle, and in a heart in no way altered in structure, *inorganic* murmurs.

Organic murmurs arise from *oscillation* (a whirling, eddy-like motion) *of the blood-current,* caused by the obstruction which embarrasses the circulation.

The physical cause of this oscillation in cases of stenosis of any of the cardiac orifices (arterial or venous) is the abnormal force with which the mass of blood is driven through the narrowed aperture; in valvular insufficiency it depends on the circumstance that a part of the blood on the further side of the affected valve regurgitates through the partially-closed opening (*e.g.*, from the left ventricle into the left auricle in mitral insufficiency, from the aorta into the left ventricle in aortic insufficiency), the backward wave thus coming into collision with the onward current; a similar movement takes place within aneurismal tumours of the aorta, the column of blood within the sac being thrown into commotion by each successive systolic blood-wave. All murmurs established in connection with disturbance of the circulation from organic causes, are thus primarily *murmurs pertaining to fluid;* unquestionably, however, these movements are often communicated also to the degenerated valves, and the murmurs are in this way greatly intensified.

Some anatomical alterations also, which do not present any hindrance to the transit of the blood, such as abundant and thick deposit on the inner surface of the ventricular walls, small tumours, &c., may occasionally give rise to murmurs; in this case, as in the others, they are to be referred to some commotion in the blood-stream.

Impediment to the circulation takes no share in the causation of *inorganic* murmurs; they are attributable solely to irregular, non-uniform vibration of the valves and arterial walls. *Uniform vibration yields sounds, non-uniform vibration, murmurs* (see p. 287).

Endocardial murmurs are always exactly synchronous with one or other of the two phases of the heart's movement, and are therefore either systolic or diastolic, or both, according as the embarrassment of circulation occurs only in the systole or in the diastole, or in both.

It is generally a matter of no difficulty to determine whether murmurs are systolic or diastolic, this being accomplished by noting the relation they bear to the cardiac impulse: systolic murmurs coincide with the apex-beat, diastolic murmurs come directly after it. But when the heart's action is very irregular (as is frequently the case in mitral valvular lesion when compensation begins to fail, and after the use of digitalis) or tumultuous, or so feeble that pulsation is no longer perceptible to the touch, the rhythm of the murmur can be ascertained by placing the finger on the carotid artery, the throb of which is synchronous with the impulse of the heart. Palpation of the radial artery is not to be trusted to for this purpose, as the pulse at the wrist is appreciably later than the heart's contraction. *Systolic* murmurs are further commonly distinguished from diastolic murmurs by being more *accentuated*, and usually also louder, as the force under the influence of which they originate is that supplied by the muscular contraction of the heart itself; *diastolic* murmurs, on the other hand, are generally *prolonged* and *not accentuated*.

Endocardial murmurs, both systolic and diastolic, are very variable in *character;* they are usually blowing or softly aspirated, but may also be of a rustling, sawing, scraping, grating, whistling, or singing quality. Not unfrequently a murmur presents several of these peculiarities simultaneously.

None of the properties just mentioned possesses any differential diagnostic signification; though the differences in the murmurs are *in part* due to the special form of degeneration in which the valves or the structures round the cardiac orifices are involved, all the various diseases to which these parts are subject set up the same hindrance to the circulation, and it is only the latter, the nature of the obstruction, that is the proper object of diagnosis.

The *intensity* of murmurs is as variable as their quality. Sometimes they are so faint and soft that they are recognized only after long and attentive examination and by eliminating the respiratory sounds (causing the patient to suspend respiration); at other times they are so loud as to be heard not only in the præcordial region but also over the whole anterior, and occasionally also the posterior surface of the chest. In the latter case they may be audible even to the patient himself, and the examiner may hear them while his ear is still a short distance from the chest-wall. Such a pitch of intensity is reached only by systolic, never by diastolic murmurs.

Amongst the conditions which influence the intensity of a murmur the most important is the *energy of the heart's action*. The more violent the whirling movement of the blood-stream at the degenerated valves or contracted orifices the louder the murmur so produced. A murmur, therefore, which is scarcely audible while the heart contracts quietly and regularly, may be transformed into one of a loud and distinctly-marked character when the action of the heart is exaggerated (as by walking rapidly or raising the arms frequently), and murmurs which before had no existence may by this means be at once developed. This sudden springing into notice under excitement is particularly characteristic of diastolic murmurs at a contracted mitral orifice, these, under ordinary circumstances, often entirely escaping the ear (see p. 289). Murmurs may also frequently be intensified by pressing firmly with the stethoscope on the surface of the chest in examining.—When the muscular power of the heart diminishes, especially from fatty degeneration of the cardiac substance, at the stage in which compensation fails, murmurs become weaker and sometimes almost completely disappear.

In those cases in which the murmur has its origin in some obstacle to the free passage of the blood the heart generally contracts with greatly increased vigour, as the right or left ventricle undergoes a certain amount of hypertrophy to enable it to overcome the difficulty with which it has to contend in keeping up the circulation. Such murmurs are accordingly usually loud. Inorganic systolic murmurs, on the contrary, not being caused by obstruction to the blood-current but merely by irregular vibration of the valves and arterial walls, conditions which never lead to hypertrophy of the heart, are almost invariably weaker than those determined by organic change.

The intensity of the murmur is by no means invariably proportionate to the *gravity* or extent of the anatomical lesion to which it is due; it not unfrequently happens that on post mortem examination very slight changes are found in the valves of those who, during life, had presented a very loud murmur, and *vice versâ*. For example, the intensity of the systolic murmur resulting from degeneration of the whole extent of the mitral valve is not necessarily greater than that generated when the valve is *still in part intact.* Only such murmurs as proceed from a constricted orifice show any correspondence in intensity with the degree of contraction present.—On the other hand, the nature of the degenerative process which has taken place in the valves and orifices occasionally, but not always, has a marked influence on the strength of the murmur; very hard excrescences excite a louder, rougher murmur than a soft deposit.—Finally, all murmurs are *louder* when the patient *stands* or *sits* than when he is recumbent; in the latter position indeed the softer murmurs are sometimes entirely suppressed.

The *duration* of a murmur is very different in different cases; in some instances it is short, in others it takes up the whole of the systole or diastole, while if both periods of the cycle be occupied by murmurs the latter may be so prolonged as to leave scarcely any appreciable pause between them.

In general organic murmurs are of considerably longer duration than the normal sounds of the heart, as the latter arise simply from the short, transient tightening of the valves, while the former are produced during the whole of the time that the whirling commotion of the blood-current lasts. This is true especially of the diastolic murmurs; the ordinary diastolic sounds are heard only at the commencement of the diastole, but the murmurs often continue to near its end; in another class of cases, however, such murmurs appear only towards the close of the diastole.

For this reason the pauses observed between the sounds of the heart have been by some subdivided, the interval which elapses between systole and diastole being designated *perisystole*, while that which comes between the diastole and the following systole is divided into two parts, of which the first is termed the *peridiastole*, the second, which shortly precedes the systole, the *presystole* (Gendrin). This classification, except for the fact that it recognises the importance of the presystole, which will be found more minutely discussed on p. 290, is of no particular practical value, as it is not on the longer or shorter *duration* of a murmur that the diagnosis of any valvular or other anatomical lesion rests, but simply on the *existence* of the murmur as the prime fact, on *the exact point in the cardiac cycle* (in the systole or in the diastole) at which it makes its appearance, and on the consecutive changes found in the heart.

These anatomical structural modifications, which consist usually of the deposition of lymph and the formation of excrescences on the valves and arterial orifices, atrophy or shrinking of these parts, adhesion of the valvular segments to each other or to the endocardial lining of the heart, &c., are always the sequelæ of a previous attack of acute or chronic endocarditis.

INORGANIC (ACCIDENTAL, ANÆMIC) MURMURS.

Whilst, as already remarked, *diastolic* murmurs appear *only* in connection with some actual mechanical disturbance of circulation, it is one of the commonest experiences to find a *systolic* murmur in a heart in which there is no valvular disease or other anatomical lesion, that is, under normal conditions so far as the organs of circulation are concerned. Such a murmur, in contradistinction to those which result from organic affection, is termed an *inorganic* murmur.

Inorganic murmurs are distinguished from those of organic origin by the following characters :

1. By their softness, feebleness, and short duration ; they are of a gently blowing quality or softly aspirated, never harsh, sawing, or rasping, &c.

2. By their rhythm ; they are, as already noted, *never diastolic*,* but invariably systolic and generally attended by a more or less marked systolic heart-sound.

3. They occur most frequently at the pulmonary orifice, next most often at the mitral orifice, but very seldom at the aortic or tricuspid valve. The systolic murmur is most usually limited to the pulmonary orifice, sometimes it is heard over both it and the mitral valve, occasionally over the latter alone, and very rarely over *both* auriculo-ventricular valves and both arterial orifices.

4. *They are very commonly*, in chlorotic subjects, *combined with murmurs in the veins of the neck.*

5. They are not permanent, but become feeble as the general health improves, and ultimately disappear altogether.

Systolic, inorganic murmurs are noticed not unfrequently in *severe acute diseases*, such as pneumonia, typhus, puerperal fever, scarlatina, small-pox, in the later stages of relapsing fever, &c.; but it is particularly in *anæmic* conditions that they are heard, whether this impoverishment of the blood be due to direct loss by hæmorrhage (as in women who have been recently confined) or to chronic disease leading to changes in the constitution of the blood, (especially *chlorosis*, pernicious anæmia, leukæmia), to marasmus (as in carcinoma of the various organs and in malarial cachexia), or occasionally to pregnancy.

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* Several authors state that in some extremely rare cases they have observed an inorganic murmur *diastolic* in rhythm ; I have never yet seen such a case.

Inorganic murmurs at the mitral orifice are caused by the unequal tension of the segments of the valve, and at the pulmonary orifice by the unequal tension of the sides of the artery.

This abnormal vibration (or tension) of the valves and arterial walls is chiefly the result of slight fatty metamorphosis of the muscular substance of the heart, more especially of the *papillary muscles*, which takes place whenever anæmia becomes profound.*

When the papillary muscles, as the consequence of fatty degeneration of their constituent fibres, contract unequally and with less energy than in health, the tension of the auriculo-ventricular valve with which they are connected is likewise diminished and non-uniform ; in this way, as in cases of dilatation of the heart, a temporary *relative insufficiency* of these valves, particularly of the mitral, may be produced.

The recognition of an inorganic murmur is, as a rule, comparatively easy, its character and seat, the general morbid condition in which it seems to have its origin, and the existence of anæmic murmurs in the veins of the neck, being usually sufficient to establish its identity. The inorganic nature of a murmur, however, is most conclusively demonstrated by proving the absence of consecutive organic changes in the heart.

As an inorganic murmur (in chlorosis, for instance) may be loudest at the apex of the heart, and as the heart's action is commonly also increased in vigour, the first impression communicated to the examiner may be that he has to deal with mitral insufficiency, but this hypothesis is at once disposed of by the *absence* of hypertrophy of the right heart and of any intensification of the second pulmonary sound. It is, nevertheless, quite conceivable that in certain cases in which chlorosis is present murmurs may be generated by a *relative* insufficiency of the mitral value.

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* In animals fatty degeneration of the heart may be experimentally produced by repeated and copious venesection (Perl).

PROPAGATION AND POINT OF ORIGIN OF CARDIAC MURMURS.

When it has been ascertained simply that a murmur, systolic, diastolic, or both, is heard over the heart, the only diagnostic inference that can be drawn is the general one that there exists some obstacle to the circulation of the blood within or near the heart; but the particular valve or arterial orifice at which it arises can be determined only by finding that it is loudest at a certain spot corresponding to the anatomical situation of that valve or orifice, by fixing, in other words, its point of *maximum intensity*. Wherever the murmur presents greatest intensity, there is the centre from which it emanates; the further the ear is removed from this spot the feebler becomes the murmur. In general murmurs are propagated to, and reach their maximum intensity at, certain points according to the same laws that are found to hold good in the case of the heart-sounds; they are transmitted most distinctly, therefore, towards those parts at which the heart is not covered by lung and in the direction taken by the blood-current. Thus, murmurs arising at the *mitral valve are loudest at the apex of the heart* or immediately above it, *tricuspid murmurs are loudest over the lower part of the sternum, pulmonary murmurs in the second left intercostal space close to the sternum*, and *aortic murmurs in the second right intercostal space at the edge of the sternum and over the whole length of the body of that bone.*

1. MITRAL MURMURS.

Systolic murmurs originating at the mitral valve, whether that structure be functionally competent or not, are almost always of maximum intensity at the apex of the heart, while at that point beneath which the valve really lies they are but faintly audible, as the heart is there sheltered behind a layer of pulmonary tissue. Even in those cases in which the base of the heart is not so covered over by lung, whether this be due to retraction of the anterior border of the lung from hypertrophy and dilatation of the right heart or to the presence of pulmonary condensation complicating the cardiac lesion (a very rare circumstance), the murmur is still loudest at the apex, the cases being very exceptional in which it is of *greatest intensity* over its point of origin, that is, in the *second left intercostal space*. The results of post mortem examination warrant us in attributing the last-mentioned phenomenon, when it does occur, to *hypertrophy of the left auricular appendix*, as this part of the auricle, when much enlarged, comes into immediate contact with the anterior wall of the chest (Naunyn); thus, as in every case of mitral insufficiency the systolic regurgitant current of blood, rushing from the left ventricle, enters not only the corresponding auricle but penetrates also to its appendix, (the cavities of both parts being continuous), the further the latter passes round the pulmonary artery and the nearer its apex comes to the anterior chest-wall, the more favourable are the conditions presented for the propagation of the mitral murmur through the left auricle into the appendix and thence to the thoracic parietes.

Naunyn based his opinion that the abnormal intensity of this murmur in the second left intercostal space is really caused by hypertrophy of the left auricular appendix on a simple experiment: he found that if at the spot at which the murmur was loudest during life a slender needle be thrust perpendicularly into the chest it pierces the appendix exactly at the part at which it turns round the pulmonary artery and comes forward towards the anterior surface of the heart.

The *diastolic* murmur arising from stenosis of the mitral orifice is always loudest at the apex of the heart and in the region immediately adjoining it towards the right; it is transmitted, therefore, in the direction of the blood-stream, as it is produced by the commotion of the latter in passing from the left auricle into the ventricle.

But this murmur, apart from the circumstance that it presents its greatest intensity at the apex, is marked by one very distinctive feature by which it may be known from the other diastolic murmurs heard over the heart, particularly from those emanating from the aortic valves: it is not of precisely the same quality throughout the whole period of its duration, but is generally divided into *two*, sometimes even *three portions*, which are not indeed separated from each other by any very appreciable pause but which differ very strikingly in character and in the rapidly increasing loudness of the murmur from its commencement to its end. Thus, while the murmur is at first feeble and soft, it speedily becomes louder and towards the end of the diastole is usually rough, rasping, or grating in quality.—Frequently, also, in cases of mitral contraction no murmur whatever is audible over the apex of the heart at the beginning of the diastole, this being developed only at the end of the diastole, immediately before the systole; it is accordingly in such circumstances named a *presystolic* murmur.

That the murmur is so often absent at the *commencement*, and is developed towards the end, of the diastole, that is, in the presystole, is probably to be explained in the following way: when the heart is at rest in the diastole the pressure on the blood flowing from the left auricle into the ventricle is very low, and if the valvular orifice be not excessively contracted and its walls not very rough the blood-stream encounters no resistance in its passage through it, and therefore no murmur is set up; but when, just before the contraction of the ventricle, in the presystole, the auricle contracts, the onward current into the ventricle is suddenly quickened, the pressure at the narrowed auriculo-ventricular opening is considerably increased, the blood is thrown into commotion and a murmur is of necessity produced.

In like manner the presystolic murmur may be intensified, and a true diastolic murmur, coming shortly before it, may be called into existence, by exciting the heart to more forcible contraction. In such cases, therefore, there is generated a murmur which occupies the *whole* of the diastole, but which, as it is added to and rendered louder in each presystole, is composed of two distinctly recognisable parts.

As stenosis of the mitral orifice is almost invariably complicated by insufficiency of the mitral valve, the diastolic-presystolic murmur generally passes at once into the systolic murmur resulting from that insufficiency; the latter is often accompanied by a short dull systolic heart-sound when the valve at any part still preserves enough of its normal structure to enable it to enter into vibration.

Sometimes in stenosis of the left auriculo-ventricular orifice the diastolic or presystolic murmur disappears entirely when the heart is acting quietly, and is replaced by a *double diastolic sound*, which is immediately converted into a diastolic murmur when the force of the heart's contraction is increased (compare p. 278).—The diastolic valvular sound also, proceeding from the arterial orifices and propagated downwards towards the apex of the heart, is commonly heard along with the diastolic or presystolic murmur.

The long duration of the diastole, which in cases of mitral stenosis is owing to the slow filling of the ventricle, explains the *long duration* *of the diastolic murmur* as compared with the *shortness* of the systolic sound or, when the stenosis is complicated by insufficiency, of the systolic murmur.

2. Aortic Murmurs.

Murmurs originating at the *aortic orifice*, at the *aortic valves*, and beyond them in the *ascending aorta* are, like the normal arterial sounds, conducted with greatest intensity in the direction taken by the blood-stream, and are therefore frequently very loud and distinct in the *second right* intercostal space at the sternal insertion of the third right costal cartilage; *they have the same intensity*, however, *over a large part of the sternum*, and are indeed often *louder over that bone* than at the point just mentioned. It is essential, therefore, in investigating any case of aortic disease, that the *whole* of the sternum should be carefully auscultated, as the murmur presents its greatest intensity sometimes at one spot, sometimes at another, on the surface of the bone.

The *systolic* aortic murmur is caused by contraction of the aortic orifice, by rough excrescences which retard the current of blood through it, by atheromatous disease in the initial portion of the vessel, and by aortic aneurisms. The *diastolic* aortic murmur arises from the regurgitation of blood from the aorta into the left ventricle when the semilunar valves which guard the aortic orifice prove incompetent; it is distinguished from the systolic aortic murmur by its longer duration, and from the diastolic mitral murmur (which, however, it resembles in point of duration) in being of more uniform character; it is not, like the mitral murmur, divided into several detached and dissimilar portions, but preserves its rushing or blowing quality in full intensity often throughout the whole of the diastole. This peculiarity alone, apart from the situation in which it is best heard, is sufficient to identify it as aortic in origin.—Systolic and diastolic murmurs may co-exist, and are then generally separated from each other by a well-defined pause, though occasionally the one runs directly into the other without any such interval.

3. Tricuspid Murmurs.

These are commonly loudest over the lower part of the sternum. As they are exceedingly rare, however, the diastolic murmur being even rarer than the systolic, and as aortic murmurs are frequently heard with great distinctness in the same region, the actual existence of a genuine tricuspid murmur can be affirmed only when the consecutive signs of aortic lesion are obviously wanting and those of tricuspid lesion (more particularly the venous pulse) are demonstrably present.

4. Pulmonary Murmurs.

Murmurs emanating from the pulmonary artery are of extreme rarity; when they occur they are most plainly audible in the second left intercostal space, close to the sternal insertion of the third rib, that is, exactly at the part beneath which the pulmonary orifice is situated.

The different heart-murmurs may be *associated* with each other in various ways. The commonest instance of this is seen in the combination of two murmurs, a systolic and a diastolic, at the same spot; this takes place most often at the mitral valve, next most frequently at the aortic orifice.—The other variety of combination is that in which *murmurs arise at two or even more separate points* within the heart. The fact that such murmurs originate independently of each other in any given case is placed beyond doubt by observing that they are of nearly *equal intensity*, and are possibly very dissimilar in timbre, at two different points on the surface. If the murmur be simply systolic at one of these and exclusively diastolic at another, it is self-evident that it is double, owing its existence to morbid alterations at more than one part of the heart. But even when two systolic or two diastolic murmurs present themselves at two points in the præcordial region the recognition of their separate origin is as a rule very easy. As tricuspid and pulmonary lesions are on the whole somewhat uncommon, the murmurs which are most often combined are those proceeding from the mitral and aortic valves.

The occurrence of two murmurs, at two of the valves or two of the orifices of the heart, does not affect their propagation or the localization of their points of greatest intensity. Thus if there be a systolic murmur at the mitral valve and a diastolic murmur at the aortic valves, the former, as already indicated, continues loudest at the apex, the latter at the sternal insertion of the third right rib or over the surface of the breast-bone.—The number of murmurs heard in the præcordial region varies with the number of valves or cardiac orifices involved in disease. At each auriculo-ventricular valve and orifice two murmurs may be generated, one, systolic in rhythm, when the valve is incompetent, one, diastolic, when the orifice is contracted; and similarly, at the root of each of the great arteries two murmurs may originate, one, systolic, when the arterial orifice is narrowed, and one, diastolic, when the valves which guard the orifice become insufficient. It is to this that the great variety of combination into which murmurs enter in complicated cardiac diseases is due. I had for a long period one case under observation, in which five different murmurs were distinguishable, one systolic and one diastolic at each of the arterial orifices and one systolic at the mitral valve.

Occasionally the differentiation, particularly as regards timbre and seat of greatest intensity, of the various murmurs dependent on cardiac diseases of a complex nature, is attended by some little difficulty, on account of the influence which these morbid sounds exert on each other in being propagated through the tissues; as a rule, however, a consideration of the other physical signs and the consecutive changes in the heart, as well as of the condition of the circulation generally, is sufficient to guide one to a safe conclusion with reference to the point at which a murmur is produced.

The following is a *résumé* of the physical signs connected with the various heart murmurs.

1. MITRAL INSUFFICIENCY.

A systolic murmur presenting its greatest intensity at the apex of the heart (the systolic sound being either completely wanting or still heard with greater or less distinctness), *hypertrophy and dilatation of the right ventricle, and intensification of the second pulmonary arterial sound.*

2. MITRAL STENOSIS.

A diastolic or presystolic murmur loudest at the apex (sometimes also, when the heart's action is tranquil, *reduplication of the diastolic sound*), *hypertrophy and dilatation of the right ventricle and intensification of the second pulmonary arterial sound.*—When, as is very frequently the case, there is mitral insufficiency as well as stenosis present, the diastolic murmur is combined with one which is systolic in rhythm.

3. TRICUSPID INSUFFICIENCY.

A systolic murmur most distinct over the lower part of the sternum, systolic *pulsation of the jugular veins*, and sometimes very marked *enfeeblement of the second pulmonary arterial sound* (from lowering of the blood-pressure within that vessel).

4. Tricuspid Stenosis.

This is an extremely rare cardiac lesion, scarcely ever occurring *alone*. It is marked by a diastolic or presystolic murmur, loudest over the lower part of the sternum, and by presystolic pulsation of the jugular veins.

5. Stenosis of the Aortic Orifice.

A systolic murmur most audible in the second right intercostal space at the sternal insertion of the third right rib, of equal intensity over the whole of the upper part of the sternum (the first sound being absent or still heard along with the murmur), *hypertrophy and dilatation of the left ventricle, a very small radial pulse.* This affection is generally complicated by insufficiency of the aortic valves, when the signs proper to the latter are added to the foregoing.

6. Aortic Insufficiency.

A diastolic murmur of maximum intensity over the greater part of the sternum and in the second right intercostal space at the sternal insertion of the third rib; the first aortic sound is often muffled, or superseded by a murmur, as the entrance to the vessel is usually roughened by structural change or deposit, and is therefore the seat of more or less considerable narrowing. In the carotid artery the first sound is commonly of a similar, indistinct character, or is replaced by a murmur, *the second carotid sound being absolutely wanting* or masked by the diastolic murmur transmitted from the aorta. This lesion is further characterised by *hypertrophy and dilatation of the left ventricle, and a bounding pulse.* The *arterial pulse* may be *accompanied by an audible sound.*

7. Stenosis of the Pulmonary Arterial Orifice.

A systolic murmur most marked in the second left intercostal space close to the sternum; hypertrophy and dilatation of the right ventricle.

8. Insufficiency of the Pulmonary Arterial Valves.

A diastolic murmur whose seat of maximum intensity is in the second left intercostal space; hypertrophy and dilatation of the right ventricle.—Pulmonary arterial lesions are very seldom met with; when they do occur both forms, stenosis and insufficiency, are most commonly combined.

Aortic Aneurisms.

These sometimes give rise merely to systolic murmurs, or, if complicated by insufficiency of the valves, also to diastolic murmurs, the systolic sound being still audible or entirely abolished.

The *point of maximum intensity* of aortic aneurismal murmurs generally coincides with that of murmurs due to aortic stenosis or insufficiency, the propagation and distribution of such murmurs, however, varying with the size and the precise situation of the tumour. Thus, the murmur from an aneurism of the ascending portion of the arch of the aorta is heard most clearly in the second right intercostal space and over the sternum, that from aneurism of the transverse part of the arch passing still further to the left of the sternum. All these murmurs are frequently perceptible to palpation over a large extent of surface. They may, however, be entirely wanting even in aneurisms of moderate size, provided that the aortic orifice be not contracted by very rough growths and that the sigmoid valves be still intact; in such circumstances simple heart-sounds of greater or less purity take the place of murmurs. Aneurisms of the aorta lead to hypertrophy of the left ventricle when, as is usually the case, the aortic valves are at the same time insufficient; but should the latter structures continue capable of effecting complete closure of the orifice the ventricle may show no increase in size.

PERICARDIAL MURMURS.

So long as the opposed visceral and parietal surfaces of the pericardium retain their normal perfect smoothness, the gliding of the one upon the other, which necessarily takes place when the heart contracts and moves about in the pericardial sac, is accomplished absolutely noiselessly. But when they are roughened by the deposit of fibrinous exudation, as the result of inflammation of the pericardium, the friction of the one on the other, due to the heart's movements, gives rise to certain murmurs, known as *pericardial friction-murmurs.*

The impression these sounds make on the ear is that of a light rubbing, scratching, grating, &c., characters which alone are generally sufficient to distinguish them from the soft blowing murmurs of endocardial origin.

Pericardial friction-murmurs also, those at least arising on the front of the heart, are very frequently accompanied by well-marked *fremitus* which, except that it is somewhat feebler, is in no way different from the thrill due to pleuritic friction. The general character of this vibration is such as to suggest a very superficial origin,—it seems to proceed from a point immediately beneath the chest-wall; as there is no palpable *endocardial* murmur which bears any resemblance to the palpable *pericardial* murmur in this particular respect, attention to this sign alone is enough to set at rest any doubt as to the origin of a murmur.

Next in diagnostic importance to this superficial quality is another by which pericardial may be known from endocardial murmurs: *they are not permanently synchronous with the systole or diastole, but are apt to occur irregularly in the cardiac cycle,* following at one time the systole, at another the diastole, according to the situation of the points, on the surface of the heart, at which the roughened pericardial membranes come into contact. Thus, a friction-sound may be heard at the base of the heart an instant earlier than in the region of the apex, as the contraction of the auricles precedes the ventricular systole.—Most commonly both of the heart-sounds remain audible, the friction-murmur being interposed between them. Pericardial murmurs, also, are never propagated to such a distance as endocardial murmurs; sometimes at parts comparatively close to those at which they are distinctly appreciable they become feeble or even disappear completely.

The *intensity* of pericardial murmurs depends principally on the following conditions: the amount and thickness of the fibrinous deposit, the degree of energy with which the heart acts, the locality at which the murmurs are generated (other things being equal, therefore, friction-sounds developed towards the front of the pericardium are louder than the others), and the attitude of the patient. Friction-murmurs may be caused to disappear from one part and reappear at another by changing the attitude of the patient and so altering the position of the heart, the visceral and parietal surfaces of the investing membrane being then brought to bear on each other at different points. In the same way they may be weakened or even abolished by placing the patient on his back, or, on the other hand, they may be first called into existence, or intensified, when he is caused to sit or stand or to turn over to the left side. Inspiration, when it coincides with the contraction of the heart, has usually the effect of increasing the intensity of the friction-murmur.

The various conditions which have been described above as diminishing or increasing the intensity of pericardial murmurs have no such influence on endocardial murmurs; a consideration of these diagnostic indications, therefore, apart from the other signs of pericarditis, usually enables one to recognise with certainty the pericardial nature of a murmur, even when the latter presents a low degree of intensity.

It is only very soft and feeble pericardial murmurs, such as communicate no feeling of vibration to the hand and thus want one of their most characteristic features, that are apt to be mistaken for those of endocardial origin; this happens particularly when the heart acts irregularly and with great rapidity, when there is almost no possibility of making a distinction between systole and diastole. But even in such cases prolonged observation, and examination when the heart's action is quiet, generally warrant the formation of a positive diagnosis. —No reliance is to be placed on the rule sometimes laid down, that simple pressure with the stethoscope, which brings the two pericardial surfaces into more intimate contact with each other, serves to distinguish between pericardial and endocardial murmurs by intensifying the former and weakening the latter, as murmurs really taking their rise within the heart are sometimes rendered louder by this manœuvre, —occasionally even a pure systolic heart-sound at the apex may, by exercising pressure in this way, be transformed into a well-marked systolic murmur.

Sometimes friction-sounds which differ from each other in some particular, but which nevertheless are of equal intensity, are heard at several points in the præcordial region; such murmurs are not produced at and transmitted from one common point of origin, but proceed from several distinct and separate spots on the heart's surface.

Friction-sounds are not usually audible throughout the whole period that the pericarditis lasts, but, as a rule, only at its beginning and end,—at the outset of the disease, as the amount of exudation poured out is still comparatively small and the two layers of the pericardium are thus permitted to come into closer relation to each other, and towards its termination, as the fluid portion of the exudation is now absorbed and only the firm fibrinous portion is left behind; but at that stage of the affection at which the exudation is most abundant the murmur disappears, the serous surfaces being held apart by the mass of fluid interposed between them. Pericardial and pleuritic friction are thus subject to exactly the same laws as regards the particular stage of the disease at which they are observed.—Pericardial friction appears earliest and most frequently at the *base of the heart,* in the neighbourhood of the great vessels, as at this part the organ is less freely movable than in its lower segment, and the pericardial surfaces are allowed to remain longer in contact; here also a much more copious exudation of fluid is required to separate the visceral from the parietal layer of the pericardium and so prevent them from rubbing on each other. On the other hand, friction-sounds often persist for weeks after the subsidence of the pericarditis, but they diminish gradually in intensity as the fibrinous deposit melts and becomes more fluid, till they disappear altogether when the surfaces regain nearly their normal smoothness or when the two portions of the membrane are glued together by adhesions.

Pericardial friction-murmurs are not dependent solely on pericarditis; they are sometimes due to tuberculosis or cancer, ecchymoses or callous thickening, of the pericardium, muscular tumours of the heart, and similar affections. It is stated also that abnormal dryness of the pericardium, in the stage of asphyxia in cholera, occasions pericardial murmurs; for my part I have never observed murmurs of this nature, though I have frequently searched for them in patients in the condition described.

Murmurs are also sometimes caused by the presence of roughness of the *outer* surface of the pericardium, when this part of the membrane rubs against the adjoining portion of the lungs or the thoracic parietes; these are termed *extrapericardial* or pneumo-pericardial murmurs, and are identical in character with the intrapericardial murmurs.—Finally, the pericardium may be perfectly smooth but may move to and fro on the roughened pleura (in cases of Pleurisy). Murmurs (pleuro-pericardial) so produced are usually enfeebled or even abolished by simply holding the breath; very rarely they persist notwithstanding that respiration is suspended. The formation of a diagnosis between intra- and extrapericardial murmurs is possible only on taking into full consideration all the other signs furnished by physical examination, though even then it cannot always be made with perfect certainty.

AUSCULTATION OF THE ARTERIES AND VEINS.

ARTERIAL SOUNDS.

As in the initial portion of the aorta, so in the great vessels springing from it, certain sounds are heard accompanying each contraction of the heart, developed partly in these arteries themselves, but for the most part conducted thither from the aorta. The only arteries which it is customary to auscultate for the purpose of investigating these sounds, are the carotid and subclavian; in certain cases, however, to be mentioned further on, vessels of much smaller calibre, such as the brachial or femoral, are examined with the same end in view. The point at which the carotid may best be auscultated is in the fossa inter-sterno-cleidomastoidea, that best adapted for the study of the subclavian sounds being in the supra- or infraclavicular fossa, towards the acromial end of the collar-bone.

If the stethoscope be placed lightly on the carotid artery, when the circulatory apparatus is in its normal state, there are heard *two* perfectly pure *sounds* accompanying each cardiac systole, separated from each other by a short pause; the first of these coincides with the expansion, the second with the contraction of the artery. The expansion of the carotid artery is synchronous with the systole of the heart, its contraction is simultaneous with the cardiac diastole.

To prevent misunderstanding I think it is better to avoid the use of the terms "systole" and "diastole" in describing the periods in which sounds and murmurs are heard in the arteries, as those words have become so intimately associated with the nomenclature of the phases of the heart's action that they at once and involuntarily recall the latter to mind; all risk of confusion in this matter may be obviated by employing the terms "contraction" and "expansion" with reference to the arteries.—It is only in large arteries, those situated close to the heart, that arterial expansion, *i.e.*, the arterial pulse, corresponds exactly with the heart's systole; in the more remote vessels the interval between the cardiac pulsation and the arterial throb may be so prolonged that the latter comes to coincide almost with the diastole of the heart or occurs as the palmar arch, as they are expanded by each successive systolic blood-wave; it disappears, however, or becomes weaker in the later stages of this affection, when the contractile power of the left ventricle begins to fail and the tension of the arteries, and the blood-pressure within them, are consequently diminished. In those instances, moreover, in which the cardiac disease consists simply of hypertrophy of the left ventricle without any valvular defect in the aorta, a sound is heard in the larger arteries (the femoral, for example), though this sound is not by any means constant in its occurrence, and even when present is feeble; so long as the aortic valves preserve their normal structure the arteries show no unusual diminution of tension during the period of contraction, the increase in their tension is therefore not so great, nor does it take place so rapidly, as in other cases in which the valves of the aorta are incompetent.

Further, a soft, faint sound is occasionally detected over the femoral artery during the period of systolic expansion, in febrile diseases, and in anæmia and chlorosis (Weil), but only when the tension of the vessel in contraction is in some way lessened, so that the difference between the tension of contraction and that of expansion is greater than in health.

It has been stated above that sounds in the large arteries near the heart are to be considered as purely physiological phenomena, and that sounds in vessels at a distance from the heart and in all the smaller arteries have a decidedly pathological signification. There is another variety of arterial sound, however, *produced artificially, by compression,* which is different in nature from both of those just described, occupying a middle position between them. If an artery of somewhat large calibre be pressed upon with the finger or the edge of the trumpet-shaped extremity of the stethoscope, so as to close it *completely* or almost completely, its expansion at the point of compression is accompanied by a sound, as the walls of the vessel, above the obstruction, are by this proceeding more forcibly stretched and rendered tense, with each cardiac pulsation,—exactly the conditions which are held to be requisite to the physiological production of the first (systolic) sound in the great vessels, the aorta and pulmonary artery. If, on the other hand, the pressure made on the vessel be not sufficient to completely arrest the flow of blood through it a murmur is heard instead of a sound, as the blood-stream is thrown into the necessary whirling or eddy-like commotion at the narrowed part of the artery.

In all cases in which a sound, of pathological or artificial origin, presents itself in arteries remote from the heart, this sound is, with very rare exceptions, *single* and is associated with the *expansion* of the vessel. O. Wolff asserts that over arteries of medium size, such as the radial or ulnar, (in very emaciated subjects of middle age, and by being careful to exercise only a certain moderate amount of pressure with the stethoscope), *three* sounds may be recognised in each cardiac cycle, following very quickly on each other and corresponding with the phenomena of *tricrotism* as demonstrated by the sphygmograph; the first of these he explains as due to the expansion of the arteries, and the two others, occurring during the period of contraction, as caused by the arterial recoil-waves. My own observations are not confirmatory of Wolff's statements, though I have repeatedly examined, with this special object in view, patients whose condition was such as to be highly favourable to the practice of arterial auscultation.—In certain cases of very marked insufficiency of the aortic valves, not merely one sound, connected with the expansion of the vessel, is audible over the femoral artery, but also a second sound, dependent on arterial contraction; the *sound* here, therefore, is *double*. Traube accounts for this second sound by the supposition that during the period of contraction the artery subsides suddenly from the state of extreme tension into which it was thrown in expansion, to a condition in which its tension is very low (as the result of the rapid draining-away of the blood in two opposite directions simultaneously, towards the heart and peripherally into the capillaries), and that in so doing it emits a sound strictly analogous to that yielded by any other tense membrane, such as a violin-string, when it is *suddenly* relaxed. There are cases, however, in which the second sound appears only some time after the relaxation of the arterial walls is completed; to these, accordingly, Traube's explanation is not applicable.—Bamberger's theory on this subject is that the second sound is caused by the returning blood-wave; when the volume and force of the latter are above the average, and the tension of the artery in this way increased, the result is a sound, but if the wave be small and the arterial tension low a murmur is produced (see p. 307). This doctrine furnishes a ready explanation, as Bamberger points out, of the fact that the double sound is observed almost exclusively in the femoral artery, as this vessel, on account of the directness and great length of its course, is better adapted for the reception and propagation of the centripetal regurgitant wave than even large arteries which after a short course break up into numerous branches. It seems, moreover, that the occurrence of a double arterial sound is not limited to cases of aortic insufficiency; Weil reports having met with this sign in two cases of stenosis of the mitral orifice.

Arterial Murmurs.

Murmurs may arise from *local* causes in the large arteries (the carotid and subclavian), or *may be transmitted to these vessels from the heart*, or *may be developed artificially in all superficial arteries by simple pressure*. Murmurs of *local origin* and artificial murmurs are *invariably synchronous* with the arterial pulse, *i.e.*, with the expansion of the artery; *transmitted* murmurs, on the other hand, may be audible both *in the period of expansion and in that of contraction of the artery*.

1. Murmurs in the carotid and subclavian are to be set down as of *local* development, when over the heart no trace of their existence can be made out. Such murmurs, always exactly coincident in time with the pulse, not unfrequently occur even while the structure of the arteries is still perfectly normal. *One* condition, however, is essential to their existence,—increased action of the heart; when by this means the artery is widely dilated, and when this expansion is effected suddenly, a murmur is excited by the oscillation of the blood-wave as it enters the widened part of the vessel. It is nevertheless conceivable that these are not merely "fluid-murmurs" but that they may also be owing to unequal tension of the *arterial walls;* thus, as it is undoubted that the expansion of the arterial walls by the pulse-wave (in the large arteries) occasions a sound, it appears not improbable that in certain circumstances, particularly when the action of the heart is accelerated and strengthened, that is, when the arteries are more quickly and more forcibly dilated, a murmur may be generated instead of a sound. As a matter of fact it is frequently observed that pure and unmistakeable sounds are transformed into indubitable murmurs when the action of the heart is increased in vigour. These local carotid and subclavian murmurs are also always attended by a more or less distinct and loud sound. And further, whilst the really arterial murmurs, so long as the circulatory apparatus continues unaltered in structure, occur only periodically,—when the heart is stimulated to more energetic contraction,—they often persist for a comparatively lengthened period when the left ventricle is the seat of a considerable degree of hypertrophy, especially the hypertrophy which is consecutive to aortic insufficiency; here are presented circumstances most favourable for the formation of arterial

murmurs,—dilatation of the arteries and extreme and rapid tension of their walls.

In the branches of the carotid also, local arterial murmurs, synchronous with the heart's systole, are heard when these vessels undergo such morbid changes as cause them to become tortuous and dilated, and when the widened portions of the arteries are continuous with others the lumen of which is normal and which are therefore narrower. In small vessels which suddenly widen at certain parts of their course, and equally in large vessels which at points are abruptly reduced in calibre, an oscillation of the particles of blood ensues, a vortiginous motion is communicated to the blood-stream, and a murmur is produced. The arterial murmurs heard in the enlarged thyroid gland (and also in the carotid and subclavian) in exophthalmic goître, are of this nature; they are to be distinguished from the venous murmurs also observed in this kind of tumour (see p. 313) by being audible only with each systole of the heart, while those emanating from the veins are continuous.

Murmurs set up locally in the subclavian and carotid arteries, coincident with the cardiac systole, are also sometimes due to pathological changes (sclerosis) of the arterial walls, particularly when these are complicated by hypertrophy of the left ventricle of the heart, or, above all, by aneurism. The cause of the murmur in these cases as in the others, is the whirling commotion imparted to the blood-current at the diseased part.

To the group of murmurs under consideration, those developed in the carotid or its ramifications, falls to be added the *encephalic murmur*, discovered by Fisher in 1833. It occurs in nearly one-half of all mammals (Steffen); it consists of a soft blowing murmur, which keeps time accurately with the heart's systole, and is heard most frequently over the great fontanelle and in its immediate vicinity (sometimes also at the small fontanelle) from the fourth month to the second year of life, or, in certain affections in which the fontanelles remain open, even as late as the sixth year. It probably arises in the manifold windings and turnings of the arteries at the base of the cranium, and is conducted thence through the brain mass to the surface. With the cerebral murmur is often combined a cardiac-systolic carotid murmur (Jurasz). Brain-murmur is destitute of any diagnostic importance; it is present in children both in health and disease.

2. Murmurs in the carotid and subclavian arteries are also very frequently of more central origin, being *propagated* into

these vessels from the orifice and initial portion of the aorta; murmurs from other parts of the heart never reach the vessels named, or if in exceptional cases they do they are of very feeble intensity. When such arterial murmurs coincide with the *diastole* of the heart the inference is unavoidable that they proceed from the aortic orifice, as in the carotid and subclavian arteries the generation of a murmur of cardiac-diastolic rhythm is impossible, the necessary physical conditions being there wanting. This explanation finds ample confirmation in the results of auscultation of the aortic orifice; the diastolic murmur caused by insufficiency of the aortic valves is observed to be loudest in this situation. But should the diastolic murmur be soft and faint in character at its starting-point it may not be recognisable even in the large arteries, in which case, of course, nothing whatever is heard in the carotid artery during the cardiac diastole.—Those carotid and subclavian murmurs also which are synchronous with the cardiac *systole* are very frequently of central origin, being directly propagated from the aortic orifice (when it is the seat of marked narrowing or is partially obstructed by very rough growths, in cases of atheromatous degeneration and of aneurism of the aorta), and this transmission of aortic systolic murmur takes place with much greater regularity than is the case with diastolic murmurs, the former being carried along with the blood-stream towards the periphery, the latter (which are caused by the regurgitation of a quantity of blood from the aorta into the left ventricle) tending rather to pass backwards towards the heart.

3. By exercising moderate pressure with the stethoscope *artificial* murmurs may be produced in the arteries,—the carotid, subclavian, femoral, and (rarely) the brachial. They give to the ear the impression of a short blowing murmur synchronous with the pulse, often of great intensity in the larger arteries, such as the carotid and subclavian. In auscultating the apices of the lungs pressure-murmurs of this kind are often heard; on relieving the pressure they disappear at once. Very slight pressure is enough to elicit them, especially when the velocity of the blood-current is increased as the result of excitement of the heart's action. The femoral artery also, on account of its proximity to the surface, is equally favourably placed with the carotid and subclavian for the development of pressure-murmur.

As already explained, artificial murmurs owe their existence to disturbance of the blood-stream (oscillation of the particles of the circulating fluid), inasmuch as the blood-wave passes from a relatively wide channel into one which, at the point of compression, is relatively narrow.

A *double murmur*, one division of which is associated with the expansion of the artery and the other with its contraction, may, but only in persons suffering from positive disease, be generated by pressing upon and nearly closing the arteries. This double murmur is observed most often in the *femoral* artery, much more seldom in the axillary, brachial, or popliteal. It was first noticed by Alvarenga da Costa, in insufficiency of the aortic valves, and has been declared by Duroziez to be absolutely pathognomonic of that valvular lesion. It is true that the double arterial murmur is a very common and characteristic sign of aortic insufficiency, occurring, in my experience, in at least one-third of the cases, but it does not appear exclusively in connection with this affection, being occasionally met with in various other morbid conditions,—a fact which Duroziez himself has pointed out; thus, Friedreich detected it in chronic endarteritis of the aorta and of the large arterial trunks, in aneurism of the aorta, in hypertrophy of the left ventricle from contraction of the kidneys, in typhoid fever, &c. The first murmur, that which is coincident with the pulse in the femoral, is, as above stated, caused by compression of the blood-wave as it travels towards the *periphery*, the second by compression of the backward wave which, in aortic insufficiency, returns towards the *heart*.

VENOUS MURMURS.

Venous murmurs occur almost exclusively in the *internal jugular vein;* when very loud, also, but only then, they may be audible in the intrathoracic venous trunks with which the jugulars communicate (the innominate veins and the superior cava), and now and then in the femoral veins. They are observed not unfrequently in perfectly healthy persons, but in them attain only a moderate degree of intensity; they are loudest in *anæmic* patients, especially in *chlorotic females*.

On auscultating low down in the hollow between the two sternal portions of the sterno-mastoid muscles, where the lower end of the internal jugular vein, a saccular dilatation known as the *bulbus*, is situated, a persistent continuous murmur is heard in anæmic subjects. This is sometimes of a soft, blowing or humming character, at other times it is loud and hissing or roaring in quality, not unlike the noise made by the wind in the chimney or passing through a crevice, and in these latter cases is generally appreciable by the patient herself; occasionally it undergoes a peculiar musical modulation, conveying the impression of a singing murmur.* The resemblance which this murmur often bears to the sound of the well-known toy the humming-top, has led to its being designated the *humming-top murmur*, the *bruit de diable* of French authors.

Venous are distinguished from arterial murmurs by not being specially connected either with the systole or the diastole of the heart; they are *continuous* murmurs. Intermittent venous murmurs very seldom come under observation, and even then they are, as will be shown, very easily differentiated from arterial murmurs.

That the humming-top murmur really arises in the cervical veins, and not, as was formerly believed, in the arteries of the neck, is proved by a number of facts which will be found set forth in detail below. Its venous origin can also be made plainly obvious by pressing firmly on the dilated lower end of the jugular vein (so as to close it) above the clavicle, when the murmur instantaneously disappears; compression of the carotid at a higher point, where venous murmurs are not heard, does not modify either its character or intensity.

The bruit de diable is produced by the *whirling, vortiginous movement of the blood in the jugular veins.* The blood flows from the relatively *narrow* jugular vein into the relatively *wide* bulb (the part at which the vessel debouches into the innominate vein) and is thus caused to sweep in a somewhat spiral course round the walls of the chamber, so that the mode of origination of the venous hum may be regarded as strictly analogous to that of murmurs in the arteries (see p. 305). This dilatation at the lower end of the vessel remains permanently wider than the upper part of the vein, as its sides are held apart by the tense cervical fascia.

The venous hum, though continuous, is not always of quite the same intensity; its variation depends chiefly on the presence or absence of such conditions as accelerate or retard the current of blood through the veins. The quicker the venous circulation the louder the murmur, the slower the current the feebler the murmur. Venous murmurs are *intensified* in the following conditions.

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* By reason of this singing character it was named by Laennec, who fell into the error of considering it an arterial murmur, the *chant des artères*. It was first traced to its true source in the veins of the neck by Dr. Ogier Ward, in 1837.

1. *By turning the head towards the opposite side.* If the murmur be audible even while the patient holds the head erect and looks straight forward, the augmentation on turning is very striking; the examiner, accordingly, frequently avails himself of this device to render louder such murmurs as are feeble and doubtful in character. The intensification of the bruit, so brought about, is due to the circumstance that on rotating the head the cervical fascia and muscles, especially the omohyoid, on the side under examination, are put on the stretch and the jugular vein is compressed and narrowed. The normal, physiological difference in the calibre of the upper part of the vein and its bulb (the latter being, as already indicated, attached on all sides, and so made incapable of undergoing any diminution in capacity) is thus rendered still greater, and the most favourable conditions are established for giving to the blood-stream that spiral direction which is requisite to the production of the venous murmur.—In the great majority of individuals this proceeding usually developes a faint murmur, sometimes even one of a considerable degree of intensity, which is quite inaudible while the head is kept erect and not turned to one or other side. Such artificial murmurs must be carefully distinguished from those which are heard when the muscles and other structures on both sides are in an equal state of tension, and which are very seldom observed in the healthy subject.

2. *By acceleration of the outflow of blood from the jugulars.* The influence of this acceleration of the blood-current is seen in the facts that the venous murmur is louder while the patient sits or stands, than when the recumbent posture is adopted, and that augmentation of the murmur is noticed also in deep inspiration, and enfeeblement of the murmur in forced expiration; inspiration favours the efflux of the venous blood, expiration retards it. When respiration is suspended for a few seconds the pressure on the intrathoracic venous trunks is increased, and the escape of the blood from the jugular veins rendered slower and more difficult, the current through the vessels is almost arrested, the condition on which the whirling movement of the blood depends is thus practically removed, and the murmur consequently becomes feebler, and under certain circumstances is nearly abolished.

The effect produced by placing the patient on his back, by forced expiration or by suspension of the breathing, is obtained more rapidly and perhaps in a more striking form by exercising pressure on the dilated lower end of the jugular vein, as above the point of compression the vessel becomes engorged with blood, and distended to such a degree that circulation through it ceases. In the same way, by pressing on the vein with the stethoscope, alternately firmly and gently, the hum may be augmented or diminished in intensity at pleasure ; it is therefore above all necessary to the detection of the less marked venous murmurs to bear as lightly as possible on the vessel.

The bruit de diable in the *right* jugular is very much *louder* than that in the left, and from two causes : in the first place, the dilatation at the central end of the vessel is greater on the right than on the left side, and is accordingly more favourable to the production of the eddy-like commotion of the blood-stream; secondly, the blood flows out of the right jugular vein faster than out of the left, as the former opens into the right innominate vein in almost a direct line, while the latter, in joining the left innominate vein, forms with it an appreciable angle; and further, the somewhat wider right innominate vein also passes in a direct line into the vena cava superior, while the left innominate crosses from the left to the right side to debouch into the same venous trunk. The venous murmur is therefore often heard on the right side as far down as the level of the first rib, in the region corresponding to the innominate vein; at the symmetrical spot on the opposite side, however, it is no longer audible. —Occasionally, nevertheless, the murmur on the left side is as loud as, or even louder than, that on the right, though why it should in these cases be of such unusual intensity is not generally very obvious.

3. *The movements of the heart* affect the intensity of the venous hum to a much less marked degree than the conditions already mentioned. In accordance with the laws of physiology it is to be expected that the murmur should be weaker during the systole of the heart, as the outflow of blood from the jugulars into the intrathoracic veins is then to some extent impeded, and louder during its diastole, as the current of blood is favoured at this period of the cardiac cycle. This diastolic intensification of the murmur is in fact generally observed when the diastole does not happen to coincide with an expiration, which would tend to neutra-

lise its influence; the systolic decrease in the loudness of the murmur, on the other hand, is not so often demonstrable. There is, nevertheless, generally noticed during the systole an *apparent augmentation* of the murmur, the latter being reinforced by the sound of the carotid pulse. On eliminating and leaving out of consideration the sensory impression raised by the arterial pulse, and concentrating the attention solely on the venous hum, it will be found that the bruit is not really intensified during the cardiac systole.

It follows from the description just given of the continuous venous murmurs that they show a constant variation in intensity, a variation, however, which is characterised by no definite rhythm, as the factors which exercise most influence on the circulation through the jugular veins (the respiratory and cardiac movements) do not always work together, but, being only occasionally coincident, are at one time favourable, at another antagonistic to each other in their operation.

Continuous venous murmurs of a certain degree of intensity are also almost always perceptible to the finger placed on the skin above the clavicle, as a distinct *thrill* (*fremissement* of the jugular veins). This vibration is not constant as regards intensity, but is often interrupted by numerous short pauses, only the more energetic of the movements of the blood-stream in the veins being then felt.

Intermittent venous murmur is of rarer occurrence than the continuous murmurs which have been described. It has the same blowing character, but is very much feebler. It appears when the velocity of the current through the jugular veins is increased, (when, therefore, one of the conditions favourable to the generation of a murmur is present), that is, during *inspiration* or during the cardiac *diastole*. When inspiration and diastole occur together the murmur becomes still louder, but when the diastole coincides with expiration the accelerating influence of the former on the venous circulation is counteracted by the retarding influence of the latter, and consequently no murmur may be heard. A diastolic venous murmur, therefore, often disappears on suspending respiration. This serves to distinguish it from a cardiac-diastolic carotid murmur, as the latter continues unabated in intensity even when the patient holds his breath; the venous murmur, moreover, has not the well-defined thus thrown into commotion is driven out through the bronchus with which the cavity communicates. Such a murmur therefore arises from substantially the same physical causes as the bruit de pot fêlé.—During the cardiac diastole the air is readmitted into the cavity, its return, however, being attended by a murmur of but very feeble intensity. The effect of suspension of the respiration on pneumo-cardiac murmurs is very variable; they are sometimes decidedly weakened, occasionally almost abolished, and at other times in no way perceptibly affected.

2. Certain *systolic murmurs* which are sometimes heard in the *pulmonary artery* when the upper part of the left lung is to some degree *contracted* and consolidated as the result of chronic pneumonic infiltration. The shrunken tissue in such cases grasps and compresses the main trunk or one of the larger branches of the pulmonary artery, so that the passage of the blood through the constricted part of the vessel gives rise to a systolic murmur. In certain circumstances also a diastolic murmur may be produced in connection with regurgitation of blood taking place in the period of contraction of the same artery.—These pulmonary murmurs are occasionally limited in distribution to a small area in the second left intercostal space, when they may also be associated with an appreciable thrill, or they may be audible over a considerable portion of the upper segment of the thorax.

Immermann has put on record a case of this kind, in which both principal divisions of the pulmonary artery and their primary subdivisions were constricted by cicatricial contraction of the lung-tissue; during life a systolic pulmonary murmur was heard over the whole upper part of the lung, both in front and behind. Similar cases have been described by Bettelheim, Heller, Aufrecht, and others.

Quincke has drawn attention to two other special causes of *systolic pulmonary murmur*: 1, an absence of due proportion between the calibre of the pulmonary artery and that of its conus arteriosus; 2, flattening of the pulmonary artery by pressure, when it comes into abnormally close relation to the chest-wall. That the first of these conditions is really an efficient cause of such murmurs has been shown by the result of at least one post mortem examination; the necessary vortiginous movement is communicated to the blood as it passes from the relatively narrow conus arteriosus into the relatively wide artery beyond. In the second class of cases the margin of the left lung is retracted, the base of the heart is no longer covered in by pulmonary tissue, and the pulmonary artery comes forward into direct contact with the front wall of the chest; as the antero-posterior diameter of the heart is increased at each systole the pulmonary artery is at the same time thrust against the framework of the thorax and flattened, whilst the conus arteriosus suffers no such change in shape when the heart contracts; the spiral direction is thus given to the blood-current, and a murmur is the result.

3. There are other *systolic murmurs* observed at *various parts of the thorax*, which cannot be traced to the main trunk of the pulmonary artery, as in the region of the pulmonary orifice both sounds are clear and pure, but which seem rather to take their rise in the larger subdivisions of the vessel, when these are the seat of a certain amount of dilatation.

Bartels has reported several cases of phthisis in which a murmur of this nature was audible, not on the diseased side of the chest, but over a large portion of the sound lung, while the heart-sounds were at all points absolutely normal. It arose presumably in dilated branches of the pulmonary artery, was intensified by expiration, and possessed generally all the distinctive characters of a genuine arterial murmur. Over pulmonary excavations, also, systolic murmurs have been heard, which proved to be due to the fact that the cavities were traversed by a branch of the pulmonary artery isolated by the breaking down of the lung-tissue (Schrötter, &c.).

In the *subclavian* artery, immediately under the clavicle, short *blowing murmurs* are sometimes observed, synchronous with the *cardiac systole*, and this notwithstanding that the circulatory apparatus may be in all respects intact and that every care is taken to press as lightly as possible with the stethoscope. They are in no way connected with those subclavian murmurs which accompany murmur in the carotid (see p. 304, *et seq.*), but are confined strictly to the parts mentioned; they are further distinguished from the group of murmurs described on the pages just referred to, by being audible in only *one* subclavian artery, not in both. They are by no means common, but occur most frequently in cases of chronic pneumonic induration of the apex of the lung. When inspiration and the systole of the heart coincide with each other these murmurs are usually rendered much louder. Their mode of origination is probably essentially the same as that of the systolic pulmonary murmur, that is, they may be attributable to compression of the subclavian artery by contracting lung-tissue, or perhaps to dragging on the arterial wall, and consequent diminution of the calibre of the vessel, by adhesions which may have been formed either between the costal and visceral layers of the pleura near the apex of the lung, or between the outer surface of the pleural sac and the artery (Friedreich).

According to many English authors (Fuller, Palmer, Richardson, and others) the subclavian murmur is a phenomenon of not unfrequent occurrence; possibly, however, they have reckoned as belonging to this class some of the above-described murmurs which are not properly speaking to be regarded as subclavian, that is, those which are not limited strictly to the subclavian artery of *one* side. The true subclavian murmur has been found by Weil only six times in 600 cases, and I, though for several years I have watched specially for this sign, have recognised it in only three cases (of phthisis); it was always capable of being greatly intensified by pressure with the stethoscope. —It is now known also not to be invariably connected with disease of the apices of the lungs, being occasionally heard when the whole respiratory apparatus is perfectly sound.

EXAMINATION OF THE ABDOMINAL ORGANS.

INSPECTION OF THE ABDOMEN.

DISEASES of the abdominal organs are generally recognisable by simple inspection only when they occasion some evident change in the volume and shape of the abdominal cavity. As this alteration in size takes place in but a few of the many abdominal diseases, and in these only under special conditions, inspection is divested of much of its importance as a method of investigation when applied to the examination of the parts within the abdomen. To ensure accuracy also the signs which it elicits should, as a rule, be checked by palpation.

The most common change observed in the size of the abdominal cavity is *enlargement*.

When this is slight the determination of its actual existence is not always an easy matter. The normal dimensions of the abdomen vary within very wide limits. In those whose customary diet is more of a vegetable than an animal nature, in those who habitually eat to excess, in drinkers, and sometimes also in individuals who are in all respects strictly temperate, distension of the bowel and undue development of fat may, as our daily experience teaches, combine to produce a degree of prominence such as is seen in other cases only as the result of considerable ascites or decided increase in the bulk of some of the abdominal organs. Nevertheless, the presence of *pathological* increase even of a less marked character is also usually indicated by other easily-appreciated signs. Thus, the morbid processes which give rise to swelling of the abdomen modify also the whole aspect of the patient; they lead to more or less emaciation and to changes in the colour of the skin (often to pallor of the surface, or to a cyanotic or yellow, jaundiced hue, according to the precise nature of the original disorder). In such cases the contrast between the bulky abdomen and the lean chest and body generally, becomes very striking.—

Not unfrequently pathological enlargement of the abdomen is accompanied by other anomalies which owe their existence to the same causes; of these may be mentioned distension of the superficial abdominal veins, œdema of the inferior extremities, &c.

Bulging of the abdomen may be *partial* or *general*. *Partial* enlargements are most commonly due to increase in size of certain of the subjacent organs,—the liver, spleen, uterus, ovaries. Undue prominence on the *right side* is usually connected with the *liver*, the exact form and outline of which can often be easily made out, when the integumentary coverings are thin and flaccid, and more especially when the parts are viewed from the side, in profile. But hepatic tumour may assume such enormous proportions, as in many cases of carcinoma, hydatid cyst, and amyloid degeneration, that the projection which it makes on the surface is no longer merely local or partial but may involve the greater part of the abdominal wall.

Very marked hypertrophy of the *spleen* (from leukæmia, amyloid degeneration, intermittent fever, &c.) elevates the tissues in the *left side*, and on lateral inspection the anterior inner border of the organ may frequently be traced; occasionally the splenic enlargement dependent on leukæmia is of such a size as to occupy almost the whole of the abdominal cavity, and to raise its walls nearly equally at all points.

Dilatation of the stomach gives rise to a uniform, somewhat oval-shaped swelling in the epigastric region, passing a little towards the left, under the ribs and so beyond the superficial boundary of the abdomen; if the dilatation be excessive, as in a case which I witnessed, the tumefaction may be general, the whole abdomen being rendered tensely prominent. If the distension, however, be not caused by air, but by some other medium, there may be no appearance of unusual projection of the parts mentioned. Occasionally in a stomach so affected peristaltic movements, spontaneous or of reflex character (excited by rubbing the epigastrium briskly with the hand), are seen travelling from left to right, towards the pylorus; by following these movements closely a truer conception of the dimensions and shape of the organ may generally be formed than is possible even by palpation and percussion,—in some cases indeed its outline becomes in this way so distinctly visible that it may almost be mapped out on the skin.—Cancer of the stomach, which affects most frequently the pyloric end, manifests itself externally, when the tumour is moderately large, as a circumscribed elevation in the epigastrium; it must be borne in mind that a similar protrusion is caused by cancer of the left lobe of the liver.

Diseases of the *intestine* yield very few diagnostic data to inspection; large fæcal accumulation in the colon, when it produces any very considerable degree of distension of the bowel, forms an elongated and often movable tumour in the right or left iliac region.

In persons of spare habit it is often possible, by simple friction of the abdominal surface, to excite or intensify the peristaltic movements of the intestine and so to bring them clearly into view. When the intestinal tube is narrowed by a stricture peristaltic movements are seen only in that portion of it which is above the contracted part, if it be tensely swollen and apparently loaded by a stagnant accumulation of intestinal contents, and not in the part beyond the obstruction.

Tumours of the omentum (carcinomatous, hydatid, &c.) sometimes attain to enormous dimensions, but are seldom so limited as to involve that structure alone; they are generally complicated by the presence of similar morbid changes in the liver and other solid organs, or they contract adhesions with these organs, the result of which is that the projection of the abdomen is not confined to the anatomical area representing the omentum.

Tumours of the uterus, physiological (*pregnancy*) and pathological (*fibromyoma*, &c.), so long as they are not of unusual size, keep generally to the middle line of the abdomen.

Tumours of the right or left *ovary* first make their appearance low down in the abdomen, in the region corresponding to the anatomical site of the organs, to one or other side of the median line. Subsequently, when they become of considerable magnitude, they may present a swelling, moderately uniform both in outline and surface, of the whole lower and upper segments of the abdomen, so that by mere inspection it is often impossible, and even with the aid of palpation it remains difficult, to determine whether they have sprung originally from the right or left ovary. As a rule the ovarian tumour is movable; when the patient therefore turns to one side it seeks the more dependent parts of the abdominal cavity, and there renders the integument still more tense than before.

Among diseases of the *kidneys*, *cancer* and *hydronephrosis* form at first smooth or irregular swellings in the lumbar region, which afterwards, on developing still further, extend to the front of the abdomen in the right or left hypochondrium, or even to parts more remote. A *dislocated* (movable) kidney is occasionally, though rarely, appreciable by inspection, when it is seen as a flattened, roundish elevation of the tissues.—The *bladder*, when distended with urine, projects in the median line of the abdomen, making there an oval-shaped tumour reaching upwards to a variable point according to the quantity of urine retained.

In the foregoing paragraphs have been discussed only the more common of the diseases of the abdominal organs which give rise to *partial*, localised swellings recognisable by inspection. A fuller consideration of all those abdominal affections which at times present similar signs would lead us into the domain of descriptive, systematic medicine, which is beyond the scope of this work. Suffice it therefore to direct attention generally to the large, encysted peritoneal exudations, to the various new formations in the different organs and tissues of the abdomen, to the cysts, extravasations of blood, abscesses, herniæ, &c., which sometimes at one part, at other times at another, reveal their presence externally as visible tumours, and whose true nature and connection with the several organs can be satisfactorily demonstrated only by means of palpation, percussion, and above all by the closest scrutiny of the general health and of the history of the origin and course of the disease,—but which, even after prolonged and careful observation, frequently remain wrapped in an obscurity which the most skilful examiner fails to penetrate.

Uniform, general intumescence of the abdomen, apart from the somewhat rare cases of great enlargement of certain of the abdominal organs (mentioned above), is produced most commonly by the presence of some *abnormal material in the peritoneal sac* (usually fluid, more seldom *gas*) or by *distension of the bowel by gas* (meteorism).

When the peritoneum contains fluid, a condition which is named *ascites*, the smooth, uniform tumefaction of the abdomen undergoes some striking modifications on changing the position of the patient: when he lies on one side the prominence lessens or disappears in the uppermost flank but becomes more marked in the parts which are undermost; in the upright position the bulging is most obvious in the lower half of the abdomen, and when decubitus is dorsal the swelling projects less forward and gains in breadth. All these alterations depend on the movements of the fluid, which invariably seeks the lowest level in the abdominal cavity.—Only when the quantity of fluid is excessive, when accordingly the abdominal parietes are at all points in a state of extreme tension, is there no variation in shape noticeable on changing the patient's attitude.—In cases of intestinal meteorism, and when the peritoneal sac is filled with gas, the uniform enlargement of the abdomen is unaffected by the posture of the patient; this therefore is a point by which these conditions may be distinguished from ascites.

In cases of very abundant peritoneal effusion the skin over the abdomen exhibits a shining, anæmic, whitish blue coloration, and at those parts where the stretching of the integuments is greatest are seen white lines, such as those which occur in pregnancy, due to the separation of the tissue elements of the corium.

In the umbilical region, and radiating from it both upwards and downwards, blue-coloured venous plexuses are often observed, which belong to the widely-dilated epigastric and mammary veins. The widening of these vessels is always a sign of engorgement of the portal vein and of the whole portal system, and an indication that on account of this venous stasis a portion of the blood which should pass through the liver finds its way by collateral branches into the superficial abdominal veins. This sign is associated chiefly with cirrhosis of the liver, being caused by the compression of the ramifications of the portal vein which takes place in that affection. Should there also be any obstacle to the return of the blood through the vena cava inferior, as from obliteration, compression by tumours, all the superficial abdominal veins, and still more those of the lower extremities, are increased in calibre; œdema is then also generally present.

Diminution in volume (*depression*) of the abdomen usually affects equally the whole of the abdominal cavity; it is not peculiar to any one disease of the organs, but is ordinarily to be regarded merely as one of the concomitant signs of the general emaciation which attends the various consumptive disorders. It is also observed when the bowel is almost or entirely empty, as from stricture of the œsophagus, cancer of the pylorus, stenosis of the upper part of the intestinal canal. Emaciation from these causes is commonly accompanied, particularly in children, by other phenomena which are equally characteristic of a lowered state of nutrition of the skin, such as loss of elasticity and free desquamation (pityriasis).

In the basilar meningitis of children a boat-shaped depression of the surface of the abdomen is generally seen, caused by the contraction of the muscular coat of the intestines, from irritation of the nervous centres which govern the movements of the bowel.

Not unfrequently the examiner may obtain important information by noticing the movements which are communicated to the contents of the abdomen by the act of respiration and the action of the heart. Thus, large tumours of the liver and spleen sink into the abdominal cavity in inspiration and rise in expiration; in the case of the liver, indeed, the whole of its sharp lower border may be distinctly discernible during respiration. The movement given to the diaphragm by the action of the heart is frequently manifest to the eye when there is a large free effusion in the peritoneal sac, the impulse passing onward through the fluid, and making itself visible as a superficial wave which appears and disappears with great rapidity.

PALPATION OF THE ABDOMEN.

THIS is a method of examination of the first importance in the investigation of abdominal diseases. It enables us to determine the size, form, consistence, and situation of the various organs, and discloses the existence of tenderness to superficial or deep pressure, the presence of any abnormal body or substance in the abdominal cavity, &c. The information so gained, it is true, is only of a general character, it reveals simply the *physical condition* of the parts and not the exact nature of the diseases by which they may be affected; but it constitutes the groundwork on which the further diagnosis rests, a more complete or particular understanding of the case in hand being possible only on taking into consideration the other symptoms presented by the patient.

In another class of cases, especially of abdominal tumour, diagnosis is rendered exceedingly difficult or becomes at best a matter of some uncertainty, when the organ from which the tumour springs undergoes alteration in form or anatomical situation, or when the starting point of the growth can no longer be positively made out. In many other diseases also, which give rise merely to physiological, functional disturbance, but to no deformity, displacement, or change in the dimensions of the organs, palpation yields no indication of any value from a diagnostic point of view.

Palpation is most conveniently practised when the patient is laid on his back or, occasionally, on one or other side; if the tension of the abdominal walls prove a serious obstacle to its proper performance the thighs should be flexed on the body, though even this expedient is not always successful in accomplishing the object intended. The great difficulty to be overcome, apart from that presented by the varying thickness of the subcutaneous layer of fat, is the tension of the abdominal muscles, particularly of the recti. The examiner should be familiar with the general anatomical relations of these muscles, the manner and direction in which their fibres are disposed, and the resistance they offer when grasped between the fingers, to guard him from error when dealing with disease.—Palpation is easiest in emaciated individuals and in women who have borne several children.

When the abdominal organs are in their normal condition the hand meets with no unusual resistance when applied to the surface; all parts of the abdomen give a uniform feeling of softness, except those corresponding to the recti muscles, and the epigastrium (from the presence of the left lobe of the liver), where a slightly greater degree of resistance is encountered. By pressing deeply in the middle line the pulse of the abdominal aorta is felt, and frequently also the vessel itself and close to it the vertebral column; even the coils of the intestine may be distinguished when the abdominal coverings are sufficiently lax.

PALPATION OF THE LIVER.

In men the presence of the liver under the margins of the ribs, when the organ is of its natural size, is indicated merely by an increase in the resistance to pressure in deep inspiration; it is only when the abdominal parietes are unusually thin and flaccid that its edge is detected with the hand. In women, especially such as have been several times pregnant, the softness and looseness of the superficial tissues facilitate the exploration of the parts beneath the arch of the ribs and permit the more ready appreciation of the inspiratory descent of the border of the liver.

The extent to which an *enlarged* liver is accessible to palpation depends on the distance to which it projects beyond the margin of the ribs into the abdominal cavity, occasionally only its sharp edge, at other times a considerable portion of its surface, coming within reach of the finger. In extreme cases of enlargement the liver may fill up a large part, sometimes even the whole of the abdomen. The greatest amount of swelling of the organ is observed in carcinomatous degeneration, the increase in size being usually slightly less in amyloid degeneration and hydatid cystic disease; the less marked degrees of enlargement occur in cases of retention of bile, passive congestion (as that due to mitral lesion), fatty infiltration and parenchymatous hepatitis.—The palpation of an enlarged liver, both of its borders and surface, presents little difficulty, as the whole extent to which the organ is increased in volume is readily detected, provided there be no unusual tension of the abdomen, such as often results from ascites.—Palpation further determines the presence or absence of sensibility or pain in the liver, the consistence of the organ, and the condition of its surface and edges.

Pressure on the surface of the liver may be painful or painless. A certain amount of *pain* to pressure is frequently associated with the swelling arising from retention of bile (in duodenal catarrh, &c.) and with all inflammatory conditions, though it is most characteristic and most severe in malignant disease. According as the cancerous nodules are scattered on the surface of the liver or are situated more deeply in its texture, the pain is elicited by applying the hand lightly or with firmness and force; it occurs spontaneously also, and is almost constant throughout the whole course of the disease.—Spontaneous paroxysmal pain of extreme severity is felt in the upper abdominal region in cholelithiasis.

Hepatic pain originating spontaneously and aggravated by pressure, is always a valuable indication of liver-disease in general, and is of service also in distinguishing between painful and painless affections. Fatty liver, amyloid liver, and hepatic hydatid growths are *painless* to palpation.

The *surface* of the enlarged liver may be perfectly smooth or rough and nodulated, the irregularities by which it is marked consisting either of slight elevations alternated with shallow depressions, or of tumours varying in size from the bulk of a pea to that of the closed fist.—The surface of the liver is smooth in all acute and chronic enlargements connected with hyperæmia, biliary engorgement, and fatty and amyloid degeneration; it is irregular when the organ contracts in the later stages of cirrhosis (granular atrophy) and in interstitial gummous hepatitis; nodulation,—the presence of small rounded elevations or tumours,—is characteristic of carcinoma, and occasionally also of hydatid disease.

If the hepatic cancer be of the diffuse variety, cancerous infiltration, the surface of the liver is free of such nodular excrescences.

In cases of hydatid disease there are often felt on the surface of the liver several smooth projections of moderate size, which offer only slight resistance to the finger; but till the stage of the disease arrives at which the surface of the organ is distinctly bulged outwards by the vesicles the diagnosis amounts merely to a probability, and depends on the careful exclusion of all other causes of hepatic enlargement.

The enlarged liver is of a variable degree of *consistence*.

In acute swellings, such as arise from inflammation or retention of bile, the organ is slightly softer to the touch than the chronic enlargements and degenerations. Among the chronic degenerations (leaving out of consideration the cirrhotic liver, which is the hardest of all, but which need not be noticed here, as it contracts to less than its normal size and is therefore not reachable by palpation) the amyloid liver is the most dense, the fatty liver being somewhat less firm. When the distinction between these two conditions is not otherwise clear the consistence as revealed by palpation may be taken as a diagnostic criterion.

Uniform enlargement of the liver causes no alteration in the general *form* of the organ; but when the tumefaction is confined chiefly to the right lobe, or more rarely to the left, the most diverse changes in shape are met with. These affect the whole mass of the liver as well as its border, though the latter *alone* may be the seat of the greatest change, the rest of the organ not being involved, as, for instance, in cases in which echinococcus-vesicles develope in the margin of the liver. These hydatid growths and cancer (sometimes also syphilis) produce, on the whole, the greatest degree of deformity of the liver, the other hepatic diseases being generally attended by uniform enlargement of the organ in all its diameters.—Whilst the breadth of the hypertrophied liver is usually easily and accurately determinable by palpation, and its breadth by percussion, only an approximate estimate of its thickness can be formed if that estimate be based simply on the amount of abdominal enlargement present; the thickness of its lower part is more easily ascertained, particularly when a portion of its concave under surface can be felt through the flaccid abdominal walls.

A peculiar sensation, known as "hepatic fremitus" was described by Briançon, and some time after him by Piorry, as characteristic of the presence of echinococci in the liver. They state that when a short sharp stroke is delivered with the finger on the echinococcus-tumour, which must have extended quite to the surface of the liver, a tremulous sensation is felt in the finger-tips of the other hand placed on the skin close to the part percussed. That under favouring conditions, *i.e.*, over a hydatid tumour of large size, forming a marked prominence on the anterior surface of the liver, such a phenomenon may be observed is undoubtedly true, but it is also certain that in the vast majority of cases it is absent. Many of the assertions made by authors regarding the occurrence of this sign must be set down to self-deception; its comparative rarity is evident from Finsen's statement that he had not once detected it in 235 cases of echinococcus-liver that had come under his notice.* It is moreover the case that this sensation, which bears a very close resemblance to fluctuation, is not peculiar to hydatid cysts; it is felt over ovarian cysts and in cases of ascites, and even in parts containing no trace of fluid,—as when there is a large deposit of fat under the soft lax skin of the abdomen.

There remains to be noticed the sense of fluctuation, yielded by *large hepatic abscesses* which reach the anterior surface of the liver,—affections which, in our northern climate, are exceedingly rare.

The mere fact that the liver can be felt projecting from under the ribs, is not always to be taken as an indication that it is enlarged; in women who lace tightly it may be pressed downwards till its edge protrudes 2½—5 ctm. beyond the margin of the ribs, or in aggravated cases descends almost as far as the umbilicus. Downward displacement of the liver also occurs when the diaphragm is depressed by pulmonary emphysema, right pleural effusion or pneumothorax. In certain rather rare cases it sinks in the cavity of the abdomen from relaxation of its suspensory ligament; when this relaxation, which may be caused by some intrinsic affection of the ligaments themselves, becomes excessive, and when to this is added other elements (such as severe labour) which tend to aggravate the original defect, the liver may hang downwards even as far as the anterior superior iliac spine. This is described as *movable* or *wandering* liver; only nine cases of this kind have hitherto been recorded.

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* I have had under observation thirteen cases of hydatid disease of the liver; amongst those were three in which the tumours had made their way to the surface of the organ, and one in which an enormous *mass of echinococcus-vesicles was discharged through the umbilicus*, but in none of them could I discover any trace of the peculiar phenomenon under discussion.

That a simple hypertrophy of the liver, when combined with a certain amount of softening of the hepatic substance, may closely simulate hydatid disease, was strikingly demonstrated in a case which I had an opportunity of seeing. The patient apparently presented the most marked signs of hydatid affection of the liver. The diagnosis seemed so clear that some time afterwards the proposal was made to puncture the cyst and evacuate its contents, but as the patient was unwilling to submit to the operation nothing was done. At the examination of his body after death absolutely no echinococci were found in the liver. The swelling was due principally to an enormous hyperplasia of the interstitial connective tissue. The cause of the deceptive sense of fluctuation which was so marked during life could not be discovered.

The differentiation of enlarged from displaced liver is effected by determining the upper border of the organ by means of percussion. If this be normal, and if the liver also extend downwards to some distance beyond the ribs, enlargement is clearly indicated.—It is not to be forgotten also that the liver may, in certain circumstances, be found to be both displaced and enlarged.

Occasionally the *gall-bladder*, fully distended with bile, presents itself as a pyriform, moderately elastic tumour, projecting slightly beyond the edge of the liver, usually distinctly visible on the surface, but still more easily recognised by palpation, offering little resistance to the touch, and at times giving to the finger an unmistakeable sensation of fluctuation. This tumour may be temporarily diminished in size by pressing upon it and causing a quantity of the bile which it contains to flow out through the excretory ducts; I observed this in a severe case of jaundice, in which the dilated gall-bladder had produced a considerable, pear-shaped elevation of the abdominal wall. The situation of a biliary tumour of this kind at the part corresponding anatomically to the position of the gall-bladder, and the severe accompanying icterus, render its recognition easy.—The gall-bladder is, in certain exceptional cases, enormously distended with serous fluid (dropsy of the gall-bladder), when it loses all trace of its original shape and may occupy a large part of the abdominal cavity.

PALPATION OF THE SPLEEN.

The spleen, so long as it retains its normal dimensions, cannot be touched with the palpating finger; it is only in full inspiration and when the abdominal coverings are unusually soft and yielding, that its anterior lower end may be felt deep in the left hypochondrium. Palpation of the spleen is best accomplished during the respiratory pause, the finger-tips being thrust in under the left costal margin as the patient draws a long deep breath. When palpation is found to be difficult or impossible in the dorsal recumbent posture the abdominal wall may be still further relaxed, and the examination thus rendered easier, by turning the patient over on his right side.

Even a slight degree of enlargement of t of a long and full inspiration, appreciable to deep palpation, though this is not always indicated by an actual advance of its lower end but sometimes only by a well-marked increase of the sense of resistance. As the organ grows larger it takes up more and more of the abdominal cavity, extending obliquely downwards and inwards, toward the median line. In accordance with the laws of gravitation the direction in which the increase in volume of the spleen takes place is almost invariably downwards, upward displacement of the diaphragm from this cause being observed only when the tumour is of exceptional magnitude.

The slighter forms of splenic enlargement are met with in the various infectious diseases, typhoid fever, typhus, pyæmia, relapsing fever, small-pox, recent syphilis, &c., and in cases of obstruction of the circulation in the portal system, caused most usually by cirrhosis of the liver and various cardiac lesions; even in these affections the spleen, though it occasionally swells to twice its natural size and often to a greater extent, very seldom emerges from under the costal arch, but is perceptible to the hand only in deep inspiration. Intermittent fever of long standing, and amyloid degeneration, give rise to still greater enlargement; it is in leukæmia, however, that the most notable amount of hypertrophy is observed. In the last-mentioned affection it is not uncommon to find a splenic tumour of such dimensions that it fills half or even more of the abdominal cavity.

Except in those cases of enormous enlargement just referred to the spleen, when hypertrophied, generally preserves its original form, the increase which it undergoes being nearly equal in all its diameters—its length, breadth, and thickness. Splenic tumours of irregular outline, bearing no resemblance to the normal shape of the spleen, are exceedingly rare. The anatomical situation of the enlarged spleen also corresponds generally with that of the organ in health; its long diameter runs parallel to the curve of the ribs, taking a more diagonal direction than the transverse diameter. The thickness of the tumour may also be approximately estimated when its concave surface can be reached with the finger.

The *notches* or *depressions* on the surface of a very much spleen may usually be distinctly felt; the swelling of a whole deepens them to such an extent that they can be made out with the greatest precision. Generally there is only one large fissure on the spleen, and one, sometimes two, rather smaller. In cases in which there is some difficulty in recognising whether the tumour is really spleen or not, these indentations become of prime importance as diagnostic signs, as they are discovered in no other variety of swelling than that consisting of the hypertrophied spleen.

All splenic tumours of any considerable magnitude, reaching downwards some distance in the abdominal cavity, are of firm *consistence;* the leukæmic, amyloid, and malarial forms are all more or less alike in this respect, or at least differ so slightly that their density offers little that is of value in a diagnostic sense. Those associated with acute diseases, on the contrary, never attain any very great size and are always of very soft consistence; in typhus indeed they are even softer than the healthy spleen.

The *surface* of all these tumours is smooth, or at most presents no marked irregularity.

Palpation of tumours of the spleen gives rise to almost no pain, or, in exceptional instances, to only a slight feeling of uneasiness.

Finally, the spleen may form in the abdomen a distinctly visible and palpable swelling without at the same time necessarily being the seat of any enlargement; it may be *dislocated* as the result of relaxation of its retaining ligaments, when it is caused to shift about from one part to another of the abdominal cavity with every change in the posture of the body, constituting what is known as *movable* or *wandering spleen.* As it commonly sinks so far that its upper end comes to be situated just under the border of the costal arch, and as, further, it lies so close to the surface that its general configuration is readily and accurately made out with the hand, the diagnosis in such a case is easy.*

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* I have seen three cases of movable spleen. In the first case, that of a man in his thirtieth year, the spleen was replaced and kept in position by means of a bandage, when the pain, which had been till that time treated in various ways without success, and which was evidently caused by disturbance of the neighbouring organs by the spleen, immediately disappeared.—In the second case, which occurred in the person of a woman 48 years of age, the dislocated spleen lay in the left iliac region, just under the soft loose abdominal coverings; its whole outline, even its notch, could be distinguished with perfect precision by palpation, and the organ could be moved about in the abdomen to a very considerable extent. The dislocation had occurred suddenly, as the result of some violent physical effort, the patient at the same time

The diagnosis of splenic tumour in general rests, as we have seen, on the indications, furnished by palpation, relative to the situation and shape of the swelling, the presence on it of one or more indentations, and on the fact that the tumour, lying under the left lower ribs, presents an area of percussion-dulness which is continuous superiorly with that of the spleen. The precise nature of the tumour is inferred from the results of further examination. It is obviously *leukæmic* if any very considerable increase in the number of the white blood-corpuscles can be shown to exist,* or may be assumed to be *amyloid* if the patient labour under any disease in which it is known that the organs are liable to this form of degeneration (destructive changes in the lungs, diseases of the bones, syphilis—in which the liver also is usually enlarged) and if, in addition, the urine is found to be albuminous (amyloid kidneys); it may be regarded as of *malarial* origin if its appearance is preceded by chronic intermittent fever, or as due to congestion in those affections in which circulation in the portal system is impeded (cirrhosis of the liver, &c.)—

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being made conscious, by her sensations, of the nature of the accident that had befallen her; the tumour occasionally gave her slight dull pain. The splenic dulness was wanting in its normal situation, the pulmonary percussion-sound merging directly into the clear tympanitic sound of the intestines.—In the third case, that of a young woman who suffered from angular and lateral curvature of the spine and who had been twice delivered of a child by instruments, the spleen lay in contact with the flaccid abdominal wall a little above the left iliac region; the displacement was supposed to have taken place after the second confinement. The tumour was movable within certain narrow limits, and its size and shape were exactly definable by palpation. The most notable fact in connection with this case was that the patient, desiring, perhaps, to become famous as a pathological marvel, caused the spleen to be totally *extirpated;* in fourteen days she had recovered from the operation. She continued in good health for three months, when she died from the effects of another operation—colporraphia posterior.

* In normal blood there are about 300 red corpuscles to every white corpuscle, and in the field of the microscope, under a power of 300 diameters, it seldom happens that more than 10 of the latter are seen scattered among the closely packed and adherent red globules. In profound leukæmia the proportion of colourless corpuscles to red rises to 1 of the former to 10, 5, or even 3 of the latter, and cases have been met with in which the number of the white globules equalled or even exceeded that of the red. Even in the less intense forms of the affection the increase of the colourless corpuscles as compared with their rarity in healthy blood strikes the eye at once.

In splenic leukæmia, *i.e.*, in those cases in which only the spleen is swollen and not the lymphatic glands, the colourless corpuscles are found to be of large size and to contain several nuclei; whilst in the lymphatic form of the disease, in which only the lymphatic glands are enlarged, and not the spleen, the white corpuscles are small and granular, with solitary, relatively large nuclei. When, as is very frequently the case, both spleen and lymphatic glands are swollen, *both* kinds of colourless corpuscles are seen side by side.—The blood required for examination may be taken from any part of the body which is most convenient, by making a slight scratch on the skin.

In certain not very numerous cases (of which I have seen four well-marked instances) a considerable enlargement of the spleen is combined with malignant swelling of the glands of the neck, axilla, &c.; this variety of splenic tumour is distinguished from that of leukæmia by the circumstance that it is not accompanied by any augmentation in the number of the colourless blood-corpuscles. This condition has been designated *splenic anæmia*, or pseudoleukæmia.—*Hydatid* disease of the spleen is rare, there being up to the present time but 26 cases recorded in medical literature. When it does occur it may give rise to great deformity and enlargement of the spleen; if the liver be simultaneously infested by echinococci, little doubt is left as to the cause of the swelling, but when the parasites are lodged in the spleen only the principal guide to the true character of the tumour is the discovery, on its surface, of a circumscribed spot of soft, elastic consistence to palpation—the echinococcus-cyst.—Other tumours (carcinoma) are developed only secondarily in the spleen.

Splenic enlargement can scarcely be mistaken for any other variety of swelling found on the left side of the body, from whatever organ or tissue it may spring, if due attention be paid to all the details of the examination.

PALPATION OF THE STOMACH AND INTESTINAL CANAL.

Palpation of the stomach enables us to detect pain or abnormal resistance to pressure in the epigastric region. Pain felt over a circumscribed area and aggravated by pressure, almost invariably indicates gastric ulcer; diffuse pain, on the other hand, occurring periodically, is observed in almost all diseases of the stomach, whether neuralgic or associated with tissue-changes in the walls of the organ, from simple catarrh to the gravest forms of malignant growth. The pain in the purely neuralgic affections comes on in paroxysms; starting from the epigastrium it radiates both to left and right and backwards towards the vertebral column, and is sometimes of a degree of severity such as is experienced in no other disease of the stomach; occasionally also, unlike the pain due to round ulcer of the stomach, it is relieved by pressure on the epigastrium.

The new formations which are found in the stomach, usually of a cancerous nature, are almost always in their advanced stages appreciable by palpation when, as is most usual, they have their seat at the pylorus; cancer of the larger curvature of the stomach, a rarer variety of the disease, may also be felt with the hand, but that of its cardiac end or of the smaller curvature is too deeply-seated for examination with the finger. They generally form tumours of greater or less size, of considerable density, painful both spontaneously and to pressure, occasionally sharply defined from the neighbouring parts, at other times extending beyond the epigastric region and then often having intimate connections with the liver. But a painful epigastric tumour, perceptible to the touch, is not necessarily of gastric origin,—it may spring from the left lobe of the liver; the diagnosis of gastric cancer therefore is warranted only on observing the other symptoms which point to disease of the stomach.—In some rare cases the tumour of the stomach is due to hyperplasia of the muscular coat of the organ (*myoma*) or to sarcoma. These swellings, though usually small, may attain such dimensions as to constitute a distinct tumour in the epigastrium.—In pathological dilatation of the stomach the extent to which its greater curvature is displaced downwards may be demonstrated by passing a sound and feeling its point through the abdominal coverings (Leube).

Diseases of the *intestines* do not frequently present signs which are appreciable by palpation.

Large, hard, *fæcal accumulations* in the intestinal canal are usually felt on the right or left side of the abdomen as irregular, movable masses; their true nature is apt to be mistaken, particularly when, as occasionally happens, they do not disappear even on the administration of strong purgatives.—In cases of stenosis of any part of the canal the peristaltic movements of the bowel may be observed on passing the hand over the abdominal wall, which in such circumstances is generally thin and emaciated, and on the surface of which the intestine stands out in elongated, rounded and firm ridges. These movements are set up by the irritation of the mass impacted above the constriction; occasionally also they become apparent spontaneously, independently of the presence of any such cause of distension of the intestine.

Of the many varieties of painful sensations, differing widely from each other in character and distribution, associated with inflammatory affections of the bowels, that most worthy of notice from the diagnostic stand-point is the *ileocæcal pain connected* *with inflammation of the cæcum, typhoid fever, and perforation of the processus vermiformis.* If in the course of the latter affection exudation takes place a semi-solid tumour is formed, tolerably sharply defined from the neighbouring parts, perceptible to superficial or deep palpation according as the amount of the exudation is great or small.

The presence of *fluid in the gastro-intestinal canal*,—in the stomach in cases of dilatation, in the bowel often as the result of simple catarrh or of Asiatic cholera (in which the intestine is frequently loaded with an enormous quantity of fluid),—is marked by the occurrence of a loud splashing, which is not only felt but is also distinctly audible, on pressing firmly and quickly on the abdomen (see p. 371). In typhoid fever this gurgling is generally limited to the ileocæcal region; the same symptom, however, similarly circumscribed, is occasionally noticed in catarrh of this part of the intestinal canal.

The principal diseases to which the *omentum* is subject are the various new formations, especially tuberculous and cancerous degeneration; the parts in which these changes have taken place are often felt through the thin abdominal wall as hard cord-like masses. Not uncommonly they give rise to tumours of greater or less size (fibroma, sarcoma, lipoma, carcinoma, hydatid and vascular growths) whose point of origin can be determined with certainty only when the neighbouring structures are not involved in the swelling; but when this is the case, when the adjoining organs are likewise enlarged and invaded by the same degenerative process, the resulting tumour may be of such enormous size and so irregular in form that it is no longer possible to discover what share each part takes in its formation.

Tumours of the *pancreas* and of the *retroperitoneal glands* may sometimes be detected by palpation. Those of the pancreas are rare, almost always of a cancerous nature, and very seldom primary or unassociated with similar changes in other organs; they form in the epigastrium hard, immovable or only slightly movable tumours, which are evidently deeply rooted in the abdominal cavity. Under favourable circumstances the connection of these growths with the pancreas can sometimes be demonstrated during life, but a confident diagnosis becomes impossible when, as in most of the cases hitherto recorded, they are merely part of a general swelling of the abdomen. This applies also to cases of enlargement of the retroperitoneal glands, which is occasionally primary, more often secondary, and developed in the course of very many of the general constitutional diseases.

Palpation in Diseases of the Peritoneum, and in cases of Accumulation of Fluid in the Peritoneal Sac.

In diffuse peritonitis, whatever be its cause, gentle pressure or even a light touch with the finger at any part of the abdomen causes the most acute pain; in circumscribed peritonitis this extreme sensibility to palpation is observed only over the area affected. Cancerous degenerations of the peritoneum are characterised by the presence of irregular nodules, always painful to pressure, which may be defined with the hand provided there is no over-distension of the abdomen with ascitic fluid; tubercular degeneration and thickening are likewise sometimes distinguishable by palpation.

The differential diagnosis between the conditions described is based, leaving out of consideration the great difference in the course of the two diseases, on the results of the examination of the other abdominal organs; cancer of the peritoneum is found to be merely one manifestation of a much more widely spread morbid process, while tuberculosis of the peritoneum, on the other hand, occurs only in the more advanced stages of pulmonary phthisis.

Large quantities of free fluid in the peritoneal sac,—which consist usually of *transuded serum,* more rarely of exudation, —give a sensation of *fluctuation* to palpation. On placing the patient on his back or in the upright position and striking with the finger of one hand on the abdomen, a sense of fluctuation is transmitted to the other hand laid flat on the abdominal wall either near or at a distance from the part percussed; a distinct wave is also usually seen to pass over the surface.—The force of this wave depends on the quantity of fluid present and the consequent tension of the abdominal parietes; if the latter be still flaccid, the amount of effusion being small, fluctuation may be quite wanting or appreciable only in the upright position, in which the fluid sinks to a lower level in the cavity and there renders tense the superficial parts. Further, if the effusion be scanty the undulatory sensation may be elicited only in the neighbourhood of the spot percussed, and not at more distant parts.—The level to which the fluid subsides may be defined with considerable precision by noting how far upwards fluctuation extends.

Encysted peritoneal exudation, hemmed in by inflammatory adhesion of the peritoneum with neighbouring organs, gives no sensation of fluctuation. Peritoneal *transudations*, not being of inflammatory origin, are not usually encysted; where, however, transuded fluid is found enclosed in this manner the adhesions by which it is confined must be the remains of some antecedent inflammatory process.

Friction-murmurs are sometimes appreciable by palpation. If, for instance, the parietal layer of the peritoneum, and that which covers a large hepatic or splenic tumour, become roughened by chronic inflammation, the opposed peritoneal surfaces come into more intimate contact with each other during the inspiratory and expiratory movements of the liver and spleen and rough friction takes place between them, capable of being considerably aggravated by pressure on the enlarged organ.* The conditions most favourable to the development of the friction-murmur are presented in carcinomatous disease of the liver, when the peritoneum over the irregular nodules with which the organ is studded is at the same time inflamed; on the other hand, this abdominal friction is absent when the tumour is chronic in its progress, there being no provocation to inflammation of the peritoneum when the enlargement of the organ is slow.—In connection with other abdominal tumours also, when they contract adhesions with the peritoneum, evidence of friction may be obtained by palpating deeply or by moving the affected organ slightly with the hand.

PALPATION OF THE URO-GENITAL APPARATUS.

The *kidneys* become accessible to palpation when they shift from their normal situation and sink deeper in the abdominal cavity (*movable* or *wandering* kidney), or when they are the seat of great enlargement,—particularly that due to *hydronephrosis.*

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* Peritoneal friction, first accurately described by Déspres, gives to the finger the same impression as pleuritic friction,—that of scratching or grating. In one case which I saw the friction-murmur was so marked that the patient himself drew my attention to it.

The movements of the heart also, as in a case observed by Emminghaus, when the contiguous peritoneal surfaces of the liver and the diaphragm are covered by rough fibrinous deposit, may give rise to a *systolic* friction-murmur.

Dislocation of the kidney, which is very seldom congenital, but most usually acquired, occurs most often on the *right* side, rarely on the left. Relaxation of the ligaments predisposes to this affection, a fact to which is to be ascribed the greater frequency of floating kidney in women after confinement; violent physical exertion is also occasionally a cause of this displacement.

The data on which the diagnosis of movable kidney rests are the presence in the abdomen of a palpable tumour having the anatomical form of the kidney, and, if further proof be desired, the absence of the renal percussion-dulness in the region in which it is usually found.

As the dislocated kidney lies comparatively close to the abdominal wall the *smoothness* of its surface and its characteristic bean-shaped outline are readily made out when it can be sufficiently fully grasped between the two hands. On account of its extreme smoothness and very free mobility it often slips from under the finger when pressed upon. The further it has wandered from its original position the more easy is it to fix it; if it has moved only from the right lumbar region into the right hypochondrium it sometimes disappears spontaneously, or on attempting to examine it with the hand, or occasionally in consequence of various movements on the part of the patient, sinking behind the hypochondrium again and passing for a time beyond the reach of the finger. Palpation of the dislocated kidney gives no pain; it is only on using considerable force that uneasy sensations are felt in the part.

Over the region which should be occupied by the kidney the sound to percussion is clearer than that obtained at the corresponding point on the opposite side; this clear sound becomes dull when the kidney is returned to its normal position.

The palpation-signs and those elicited by percussion are as a rule sufficient to prevent a movable kidney being mistaken for an abdominal tumour. It is only in those cases in which, by pressing upon and irritating the neighbouring tissues, it has excited inflammation and thickening of the surrounding parts, as in two cases which I observed, that the diagnosis presents any difficulty, as the organ is deprived of its mobility and its distinctive outline is then no longer recognisable. In such circumstances the objective examination must be supplemented by a careful consideration of the history of the development of the disease.

If the kidneys undergo extensive degenerative changes and at the same time increase so much in size that they may be appropriately described as large renal *tumours*, they may come near enough to the surface to be examined satisfactorily by palpation. Hydronephrosis, due to the presence of some mechanical impediment to the flow of urine into the bladder, constitutes a swelling of this character. If this obstruction, arising from compression, adhesion, or malformation of the part at fault, affects only one of the ureters the hydronephrosis involves only *one* kidney, but if both ureters become impassable, or if the obstacle be situated at the urethral orifice of the bladder (cases which are somewhat rare), bilateral hydronephrosis results. The largest tumours are seen in hydronephrosis on one side; double hydronephrosis ends too rapidly in death to permit of the development of swellings of great size, while in the unilateral form of the disease the discharge of the urine secreted by the other kidney goes on unhindered.

The tumour produced by hydronephrosis, when it is of considerable magnitude, is felt both posteriorly in the lumbar region and in front through the abdominal walls, as a soft fluctuant mass of somewhat spherical shape. Its volume may lead to its being taken for an ovarian growth, but the history of the manner in which the tumour was developed and the data furnished by further physical examination, usually render such an error in diagnosis impossible.

An enormously distended *bladder*, (which has been known to reach as high as the umbilicus), is recognised by inspection, and still more positively by palpation, as a firm, elastic, oval-shaped tumour in the lower part of the abdomen.

Hypertrophy of the prostate may be diagnosed by palpation through the rectum.

Among the diseases of the female *genital apparatus*, tumours of the ovaries and of the uterus may usually be made out with the greatest ease by palpation. Ovarian tumours are generally unilateral, frequently irregularly spherical in shape, and movable. In most cases they contain a large quantity of fluid, and yield then a distinct feeling of fluctuation to firm pressure (ovarian *cysts*). In other less common cases they appear to the touch to be solid throughout; they may, nevertheless, enclose fluid, fluctuation being wanting merely because the force exerted by the hand in palpating through the thick coverings of the cyst is insufficient to set its fluid contents in motion. In multilocular ovarian cysts also there is no fluctuation. On the other hand, ovarian tumours have been observed which, during life, gave very distinctly the sensation of fluctuation, but which at post mortem examination were found to contain no fluid; they were exceedingly soft and flabby in consistence, their whole tissue being infiltrated with serum.—There is, in general, no difficulty in ascertaining whether the tumour springs from the right or the left ovary, even when it is so large as to pass to some distance over the middle line; but if from the enormous bulk of the tumour, which may fill up both sides of the abdomen equally, it becomes impossible to make this distinction by physical examination, the patient's recollections as to the part in which the swelling first appeared will usually afford trustworthy enough indications as to its starting point.

The history of the case, and especially examination per vaginam, by which latter means the growth can always be reached with the finger, will generally serve to distinguish ovarian from other forms of abdominal enlargement; the diagnosis of hydrovarium from ascites may be made with a like degree of certainty by combining palpation with percussion and by giving due weight to the history of the course of the disease, to the results of the exploration of the other organs, &c. (see p. 363).—It may seem to some impossible to confound an ovarian tumour with a gravid uterus, yet this has often been done.

Enlargement of the *uterus*,—physiological in pregnancy, pathological in cases of tumour of the uterine substance or of accumulation of fluid in its cavity,—are appreciable by palpation through the abdominal wall as soon as the organ emerges from the pelvis and rises into the abdomen.

PERCUSSION OF THE ABDOMEN.

Percussion of the abdomen is most conveniently practised when the patient is made to lie on his back, in which position the abdominal coverings, especially the recti muscles, are relaxed to the greatest extent.

The object to which percussion is here directed is the delimitation of certain of the subjacent organs,—the liver, spleen, and gastro-intestinal canal, very rarely the kidneys. The pancreas, ovaries, and uterus, when of their *normal* size, are not definable by percussion.

The diagnostic importance of percussion in many abdominal diseases is merely secondary, particularly in cases of enlargement of the various organs, the information obtained by palpation being more reliable, inasmuch as by the latter means we are enabled to detect not only increase in volume but also many other alterations to which the parts are subject and thus to form a clearer conception of the precise nature of the morbid process going on; this is exemplified in cases of palpable tumour of the liver, spleen, ovaries, uterus, and in various other abdominal tumours. In other cases, however, in which palpation gives negative or uncertain indications, percussion comes to be of the greatest value as a method of physical examination, as, for instance, in free ascites, encysted peritoneal exudation, intestinal meteorism, accumulation of gas in the peritoneal sac, atrophies and dislocations of the liver, and in the slighter (and therefore non-palpable) forms of enlargement of the liver and of the spleen. Further details having a bearing on this subject will be given in discussing the percussion of the different organs.

It is immaterial in what order the abdominal organs are percussed, though it is customary to begin with the liver and spleen and afterwards to turn the attention to the gastro-intestinal canal.

PERCUSSION OF THE LIVER.

This is undertaken with the view of ascertaining the position and size of the liver.

In normal circumstances, in which the liver is not definable by palpation, percussion is the only method by means of which the dimensions and situation of the organ can be determined, while in those cases in which, on account of enlargement, the liver projects from under the costal arch and may therefore be distinctly felt, percussion supplements the results obtained by palpation by fixing the *upper hepatic boundary;* diminution in the size of the organ can be recognised only by percussion.

The liver lies in the greater part of its extent close against the thoracic and abdominal parietes, only its upper convex surface, occupying the concavity of the diaphragm, being by the latter structure, and by the lung towards the right, separated from the chest-wall. That portion of it which is in contact with the thoracic and abdominal wall, extending on the front of the chest from the sixth rib superiorly to the margin of the arch of the ribs inferiorly, and reaching in the median line to midway between the base of the xiphoid cartilage and the umbilicus, gives at all points a dull sound to percussion. This area of dulness is known as the *absolute hepatic dulness.* That part of the liver which is separated from the chest-wall by lung-tissue extends upwards, when the diaphragm is in a medium state of contraction (as in quiet expiration), to the level of the fifth rib between the right mammillary and parasternal lines; from this point downwards to the inferior border of the lung, beyond which the liver is in immediate contact with the thorax, the sound is not dull, but is only less loud than that elicited higher up (at parts behind which lung *alone* is situated), and is therefore spoken of as merely *relatively dull.* The height of this relative hepatic dulness, however, is less than the actual height of the upper convex segment of the liver which is sheltered behind pulmonary tissue, as the dulness always begins in the middle of the fifth intercostal space, where the stratum of lung which comes in front of the liver becomes sufficiently thin, while above this level the layer of lung is too deep to permit of any diminution in the intensity of the percussion-sound taking place.

As, accordingly, the relative hepatic dulness can never be defined with rigorous exactness no attempt is as a rule made to do so, attention being directed to the determination of the area of absolute dulness only. The full vertical diameter of the organ may nevertheless be approximately shown by placing the upper boundary of the absolute dulness 4 ctm. higher,—the extent to which the liver, in the mammillary line, is covered by lung, the diaphragm occupying a middle position between relaxation and extreme contraction.

The liver should be percussed during the respiratory pause, and generally along four lines, the axillary, mammillary, parasternal and median lines.

The *upper boundary* of the hepatic dulness is found at the eighth rib in the *axillary line*, at the upper (sometimes the lower) border of the sixth rib in the *mammillary line*, at the upper border of the sixth rib (sometimes in the fifth intercostal space) in the *parasternal line*, and at the base of the xiphoid process in the *median* line. Posteriorly the liver rises as high as the level of the tenth rib. The superior boundary of the hepatic dulness is thus conterminous with the inferior margin of the right lung.

The *lower limit* of the hepatic dulness is situated between the tenth and eleventh ribs in the axillary line; in the mammillary and parasternal lines it comes close to the edge of the arch of the ribs, comparatively seldom passing beyond it, and then only to the extent of 1—1½ ctm. In women this inferior boundary may be considerably lower,—as much as 2½—5 ctm., a condition which is commonly due to tight lacing, sometimes to relaxation of the suspensory ligament of the liver; in the latter case the upper limit also is lowered. In the median line the hepatic dulness reaches downwards to nearly midway between the base of the xiphoid process and the umbilicus; from the median line it extends about 5 ctm., at most 6—7 ctm., to the left, where its lower edge, turning upwards, passes into the lower margin of the cardiac dulness and there ends, as might be expected from the anatomical site and outline of the liver, in the upper border of the hepatic dulness.—Posteriorly the lower boundary of the liver-dulness is no longer demonstrable beyond the scapular line, in which it is situated at the level of the eleventh rib; the dulness is here undefinable, being lost in that arising from the thick mass of the dorsal muscles.

The percussion-sound is not equally dull at all parts of the hepatic area; over the thick right lobe of the liver the non-resonance is much more decided than over the thin left lobe, while towards the lower margin of the organ, where it diminishes rapidly in thickness, the sound becomes more and more clear. The proximity of the intestine to the lower part of the right lobe, and of the stomach and bowel to the whole left lobe, adds to the dull sound heard on percussing in these regions one of tympanitic quality; by striking gently with the hammer or finger the confusion arising from the mingling of these sounds may be reduced to a minimum. It is only in children that, notwithstanding the employment of a light stroke in percussing, the dull sound over the whole hepatic area is accompanied by a tympanitic sound.

DISLOCATION OF THE LIVER.

In this affection the boundaries of the area of liver-dulness are altered, but the organ itself remains of normal size.

Dislocation of the liver from *physiological* causes occurs during the respiratory act; in quiet respiration the amount of displacement is merely trifling; deep inspiration, on the other hand, occasions a very considerable sinking of the liver, and consequently also of the upper limit of the hepatic dulness. At the same time the vertical diameter of the dull area diminishes, as in inspiration the lower margin of the liver descends only to a point about 1—1½ ctm. below the edge of the costal arch, whilst the upper convex surface of the organ is overspread by the expanding lung to the extent of 2—3 ctm. The attitude of the body has also a certain influence on the position of the lower border of the liver; in lying on the right side the right lobe sinks and the left rises somewhat, and if decubitus be on the other side these relations are usually reversed.

Pathological displacement of the liver takes place generally *downwards*, seldom (and then only to a very inconsiderable degree) upwards or laterally.

Dislocation *downwards* is produced by all those conditions in which depression of the diaphragm is a marked symptom,—the severer forms of *pulmonary emphysema*, right *pleuritic exudation* by palpation, especially if there be much tension of the abdominal wall; in cases of this kind the increase is clearly enough shown by the dull sound elicited by percussion of the hepatic area. This sound is never absolutely non-resonant, but is of a dull tympanitic quality. The distance downwards to which the liver reaches is indicated by the line of demarcation between the dull tympanitic sound of the organ itself and the clear tympanitic note of the adjoining intestine.

But the liver, even when distinctly enlarged and projecting below the ribs, is not demonstrable by percussion in the presence of copious effusion into the peritoneum (ascites) or meteorism of the intestine. In the first case, provided that the fluid be abundant enough to reach to the upper part of the abdomen and that the abdominal walls be in a condition of considerable tension, the dull sound of the liver is indistinguishable from that of the effusion; the fluid often rises as high as the liver, or even covers it, when the patient lies on his back. In the second case (meteorism) the colon, distended by gas, sometimes comes between the liver and the abdominal wall, when percussion educes only the loud intestinal sound. In the same way an enlarged left lobe of the liver may be so masked by the dilated, air-distended stomach as to be undiscoverable by percussion.

Of the complications just enumerated, which hinder or render impossible the delimitation of the liver by percussion, ascites is the most common (as in cases of hepatic enlargement, and peritoneal effusion in the stage of failure of compensation in mitral lesions). If the superficial tissues be sufficiently lax the swollen organ is *accessible to palpation*, when the enlargement is easily detected without the aid of percussion. Even when the abdominal parietes are moderately tense the hypertrophied liver may be explored by palpation, by pressing suddenly and firmly with the hand over that part of the abdominal surface corresponding to the site of the liver, and in this manner pushing the fluid aside.

A liver of normal size may appear enlarged when close to its lower border is situated some solid medium giving a dull sound to percussion. The hepatic dulness is thus apparently increased by the presence of firm, hard fæcal masses filling up the transverse colon, by tumours of the pylorus and of the stomach in general, and by peritoneal exudation encysted in the neighbourhood of the liver.

In these cases also, in which percussion is plainly not to be relied on as a means of investigation, palpation very generally, though not invariably, furnishes such indications as warrant a positive diagnosis. But even with the aid of the latter method of examination it is not unfrequently impossible to distinguish between cancer of the pylorus and cancer of the left lobe of the liver; it is then necessary to fall back on the other symptoms which point to the presence or absence of functional disorder of the stomach.

Several of the above-named conditions, which complicate or render uncertain the results of percussion, such as distension of the stomach by food, the overlapping of the enlarged liver by coils of intestine, the accumulation of a quantity of hard fæcal matter in the colon, &c., are of short duration, so that repeated examination usually removes any doubt that may have at first arisen as to the real dimensions of the liver; in all such cases, however, the symptoms relating to the development and course of the disease possess a much higher diagnostic value than the doubtful objective results of physical examination.

In a rarer class of cases the liver increases in size not merely downwards but also upwards, pushing the diaphragm before it, rising sometimes as far as the third intercostal space, compressing the lung and dilating considerably the right half of the chest, particularly laterally; this is observed specially in *hydatid disease of the liver.*

At the first glance such a case may readily be mistaken for one of pleuritic exudation, but a closer examination will reveal the true nature of the affection. Thus, in a woman whom I lately had under observation, suffering from hydatids of the liver, the tumour, which was large and distinctly fluctuant, was not simply abdominal but grew upwards also into the thoracic cavity, and that so quickly that in a short time the right side of the chest, from about the fourth rib downwards, was dilated to an extent such as is generally noticed only in cases of enormous pleuritic exudation. To fall into the error of confounding such an affection with pleuritic exudation is scarcely possible; apart from the objectively-demonstrable fact that the liver forms a prominent tumour in the abdomen, it will be found that the intercostal spaces, when the chest is dilated by an hepatic tumour, are not obliterated; that, further, the dilatation of the thorax is very irregular, and that above all the outline of the area of dulness does not in the least resemble that which is so distinctive of pleuritic exudation.

Contraction of the Liver.

The principal sign which this condition gives to percussion is *diminution in the extent,* or even *entire disappearance,* of the sion varies a little, however, according as the person under examination assumes the one or the other position. When he reclines on the right side the splenic dulness is displaced and slightly diminished in area; it *shifts*, as the spleen sinks and is turned still further forward (inward) at its lower end, in such a way that the direction taken by its long diameter is no longer diagonal, as in the standing posture, but more nearly horizontal; it is then also *diminished in area*, by the descent (2—3 ctm. in extent) of the lower margin of the left lung, whereby a larger part of the upper (posterior) end of the spleen is hidden by pulmonary tissue, the lower (anterior) end of the organ falling downwards. This displacement and diminution in size of the splenic dulness is recognised by comparing the areas of dulness obtained in the upright and right lateral positions; the difference is often quite perceptible even while the spleen is normal in volume, but is much more obvious when it is enlarged. It is in almost every instance advisable to percuss in *both* positions alternately, as a comparison of the outline of the non-resonant areas provides a convenient test of the accuracy of the results of this method of investigation.

The spleen, being of comparatively small size, only 2½—4 ctm. in thickness, and surrounded on almost all sides by air-containing organs, gives a sound to percussion which is much less dull than that of the liver. In adults frequently, in children generally, the splenic sound is not absolutely dull, but merely slightly muffled and at the same time of tympanitic quality; the percussion-stroke employed must therefore be gentle, otherwise vibration will be set up in the adjacent structures (the lungs, stomach, and colon), and the muffled splenic sound will be almost lost in the clear sound so awakened. The stroke must also be delivered in the respiratory pause, as during inspiration the greater part of the upper end of the spleen is sheltered behind pulmonary tissue; a very deep and full inspiration may even cause the splenic dulness to disappear entirely. Even after complete expiration, however, nearly the whole upper third of the organ, about 4 ctm. in length, remains covered by lung; this portion of the spleen is accordingly not definable by percussion,—though exactly the contrary has been maintained by some authors. At that part of the posterior thoracic surface which coincides with this upper segment of the spleen, that is, in the space lying between the ninth and tenth dorsal vertebræ and the scapular line, no diminution in the intensity of the percussion-sound is heard, the note being uniformly clear quite up to the lower border of the lung. As the upper third of the spleen is thus too deeply seated to be recognisable by percussion, it follows naturally that the long diameter of the organ as indicated by this method of examination is one-third shorter than it really is anatomically.

In percussing from above downwards—preferably without the hammer—between the left middle and posterior axillary lines, the patient being in the standing posture and having his left arm slightly raised, distinct dulness is first obtained usually at the *upper border of the ninth rib*, and at the same time the sense of resistance experienced in the finger is increased; this marks the superior boundary of the splenic dulness. This area of non-resonance extends downwards to the eleventh rib or to its lower border, which is therefore regarded as the inferior boundary of the splenic dulness; the muffling of the percussion-sound ceases at this point, and is replaced by the clear note yielded by the bowel. Posteriorly the splenic dulness is bounded by the scapular line, from the ninth to the eleventh rib; its anterior limit coincides almost exactly with the middle axillary line, also between the ninth and eleventh ribs.—The long diameter of the splenic dulness measures about 7—8 ctm., its greatest breadth 5—6 ctm., while the actual length of the spleen itself is on the average about 12 ctm., and its breadth about 8 ctm.

Whilst the spleen is easily definable between the ninth and eleventh ribs in the axillary line, the delimitation of its anterior and posterior borders in their *whole* extent is an undertaking of much greater difficulty, and one which sometimes cannot be satisfactorily accomplished. This is accounted for by the anatomical relations of the organ. Thus, the upper part of its *posterior* border lies deeply sunk behind lung-tissue, and is therefore not traceable by percussion; the middle and a portion of the lower part of the same border, also, run up to and overlap the convex edge of the kidney, so that in this direction the splenic dulness is continuous with and indistinguishable from the renal dulness. There remain accordingly of the posterior border only two small portions, an upper and a lower, which may be isolated by percussion from the adjoining resonant pulmonary area. These points being found they should be connected by a slightly curved line, which will include and indicate with substantial accuracy those other points, lying between the two extremities of the line, which are not distinctly demonstrable by percussion. The *anterior* border of the spleen, covered, like the posterior border, by lung in its upper third, comes in its lower two-thirds into somewhat close proximity to the greater curvature of the stomach, while its lowest third is in immediate relation with the splenic flexure of the colon. If the stomach be dilated, as after a hearty meal, the tympanitic or ringing metallic sound which it then gives modifies the dulness not merely of the anterior part but of nearly the whole of the splenic area. The colon, also, when distended with gas, encroaches on the splenic dulness.

The changes in form and dimensions which the splenic dulness undergoes may be caused by dislocation of the spleen, by apparent and by actual enlargement of the splenic area.

Very deep respiration is attended by a slight *dislocation* of the spleen. In inspiration the organ sinks somewhat in the abdominal cavity, but the space which it renders dull to percussion becomes smaller, as its upper end is more fully shut in by pulmonary tissue. In tranquil inspiration, however, no downward displacement of the lower limit of the spleen is noticeable. The spleen is pushed still further from its normal situation, and its area of dulness to a similar extent reduced or even caused to disappear, by left pleuritic effusion, pneumothorax, and pulmonary emphysema. In cases of pleurisy in which the fluid is so abundant as to occupy the front as well as the back part of the pleural sac, the splenic dulness and that due to the presence of the effusion cannot be defined from each other; if the exudation be unusually copious, and the diaphragm be depressed, the spleen also is displaced and protrudes from under the costal arch.—In left pneumothorax the splenic dulness may vanish completely; the spleen, however, is commonly driven downwards by the weight of the pleural effusion which is always poured out soon after the air has gained access to the pleura.—In like manner the pressure of an emphysematous lung causes the spleen to sink to a lower point in the abdomen. In a case of this kind the splenic dulness is always greatly diminished in size, as the margin of the lung creeps downward over the upper part of the spleen to a considerable distance, whilst the actual descent of the organ, due to depression of the diaphragm, is comparatively trifling.—If the spleen be dislocated upwards, as from ascites, its area of dulness is not traceable on the surface; if the displacement be the result of an extreme degree of meteorism, the splenic dulness is invaded and lessened in size, as on the one hand a larger portion of the spleen is covered by lung, and on the other the dull sound it renders to percussion is overborne by the loud and clear intestinal note.—If the dislocation be complete, if the spleen have, in other words, become *movable*, percussion-dulness is entirely wanting in the region corresponding to the normal anatomical situation of the organ.

Apparent enlargement of the splenic dulness is produced when the hollow or air-containing organs in its immediate vicinity are from any cause transformed into solid, airless structures. The dulness of these solid organs then becomes continuous with that of the spleen. This is what takes place in cases of effusion confined to the posterior lower part of the pleural cavity, consolidation of the lower lobe of the left lung, engorgement of the colon with fæcal masses, tumours of the liver of such dimensions that they reach over to the spleen, &c. Careful examination, however, will suffice to establish the diagnosis of apparent enlargement of the splenic dulness from actual increase in the volume of the spleen itself, the former condition being characterised by an irregularity of form which is strikingly different from its normal oval or rhomboidal outline, and by the total absence of the usual shifting of the area of dulness on altering the attitude of the patient, as on changing from the upright to the recumbent posture on the right side. The other methods of physical examination also at once suggest the merely apparent character of the augmentation of the splenic dulness and indicate its special causes.

Actual enlargement of the splenic dulness is the result of enlargement of the spleen itself. The non-resonant area is normal as regards shape, but is increased in size in all directions; the lines therefore which are represented above as bounding the normal splenic dulness are set more widely apart, on account of the lengthening of the vertical and transverse diameters of the splenic area. The extension of the dulness takes place most commonly downwards and backwards, when the swelling of the spleen is moderate in degree; when this is still greater the organ passes beyond its normal limits anteriorly as well as in the other directions, and when the tumour is of unusual dimensions it pushes the diaphragm upwards* before it into the thoracic cavity, and percussion then shows that the upper boundary of the spleen occupies a much higher position than normally.

As enlargement of the spleen is invariably attended by increase in the thickness (the antero-posterior diameter) of the organ, the *intensity* of the dulness is also always much greater than over the normal spleen; the percussion-sound of a large splenic tumour is as absolutely dull as that heard over the liver, while the sense of resistance experienced in the percussing finger is augmented to a very unusual degree. The larger the spleen becomes in all its diameters the more easily definable is it by percussion, the influence of the sounds given by the parts in the vicinity being eventually completely neutralised as the hypertrophy of the organ goes on.

If the spleen forms a distinct tumour, protruding from under the arch of the ribs, it must, except in some rare cases of dislocation (movable or wandering spleen), be regarded as enlarged. When it comes down in this way into the abdomen examination by percussion may almost be said to be superfluous, as much clearer and more definite information may then be obtained by palpation. The signs which a splenic tumour gives to palpation are so distinctive as very seldom to leave any doubt as to the real nature of the swelling under consideration (see p. 328). As a confirmatory test, however, percussion is here of some importance, as it shows that the line of dulness is unbroken between the normal splenic area in the lower thoracic region and the non-resonant space corresponding to the tumour.

Tumours of the spleen may be unrecognisable by percussion, or recognisable only with difficulty, if there be much meteorism or ascites present. If the swelling reach to a point considerably below the level of the ribs, however, it may always be determined, if not by percussion, at least by palpation, especially if pressure be made with the hand firmly and suddenly, in such a way as to displace the fluid for a moment.

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* In one case which I observed, in which the whole abdomen was filled by an enormous splenic tumour, the apex of the heart was found beating at the level of the fourth rib; immediately below this point also the absolute splenic dulness began.

PERCUSSION OF THE STOMACH.

A small portion of the anterior surface of the stomach, and the larger part of its great curvature, are in direct contact with the abdominal parietes anteriorly; the rest of the organ is covered over, partly by the left lobe of the liver and partly by the left lung. This portion of the stomach, that which in front touches the wall of the abdomen, is bounded towards the right by the left lobe of the liver, and superiorly by the lower border of the left lung, which turns backwards behind the sixth and seventh ribs, to the outside of the apex of the heart; the lower border of the stomach, its larger curvature, crosses the epigastrium from the left lobe of the liver to the left hypochondrium in a curved line situated nearly midway between the point of the xiphoid process and the umbilicus, inclines upwards and outwards opposite the free end of the tenth rib, and at the level of the sixth rib, in the anterior axillary line, meets the upper boundary of the organ. These are the limits of the stomach when it is in a state of only moderate distension.

It follows from what has just been stated that it is never possible to delimit the stomach in its whole circumference by means of percussion, but only so much of it as is not overlapped by other organs and is in immediate relation with the anterior abdominal wall. In examining the stomach the patient should be placed on his back, this being the most convenient position for percussion in this region. The upper limit of the stomach should first be found, the line along which the clear pulmonary note gives place to one which is tympanitic in quality; the boundary of the gastric area towards the right may then be determined by following the edge of the hepatic dulness till it ends in the lower margin of the cardiac dulness; the delimitation of the superficial gastric area is completed by tracing the course of its lower border, which coincides with that of the greater curvature of the stomach (see above). The close proximity of the transverse colon, however, often proves a serious obstacle to the proper carrying-out of this proceeding, as the percussion-sounds of the great intestine and the stomach are sometimes exactly alike both in intensity and pitch, when all that can be accomplished is usually merely to fix certain points in the line of the lower boundary. In other cases, however, when the sound of the colon is higher in pitch than that of the stomach, the course of the larger curvature may be made out with much greater precision. The percussion-sound in the gastric area just described is generally loud, clear, tympanitic, and *of low pitch*.

The gastric percussion-sound undergoes many modifications both in quality and in the extent of surface over which it is audible, according as the stomach is empty, partly filled, or completely and fully distended by its contents.

When the stomach is empty a deep and clear tympanitic note is heard over its whole extent; but if it be so fully occupied by fluid and solid matters that but little space is left in it for air, the region of tympanicity is diminished in size, and at the dependent parts of the great curvature the sound is often muffled, when it becomes easy to distinguish it from the tympanitic sound of the transverse colon, provided also that this part of the intestinal canal is not in a similarly overloaded condition. If the stomach contain only a moderate quantity of solid matters there is no muffling of the percussion-sound; if there be also a considerable volume of air present the sound may even be tolerably clear, so much so as to become indistinguishable from that of the colon.—When the stomach is overcharged with food the tympanitic gastric area is also considerably altered in extent, the above-described lower boundary-line, representing the larger curvature, being shifted further downwards, to a greater distance from the upper limit of the tympanicity proper to the stomach (the sixth rib); the somewhat crescentic gastric area is thus increased in diameter, it includes the whole left hypochondrium, overspreads and masks the anterior part of the splenic dulness and to some degree also that part of the hepatic dulness which marks the left lobe of the liver.—If the stomach be widely dilated by gas, so that its walls are in a state of considerable tension, a *metallic* quality is noticeable in the percussion-note over a greater or less area, when the ear is held close to the spot percussed or when instead of the hammer a small metallic rod is used (see p. 106). Were it possible to elicit this metallic sound over the whole of the gastric area, we should have in this system of percussing with a small rod of metal a means of fixing the boundaries of the stomach with almost absolute exactness. This method of examination has been elaborated particularly by

Leichtenstern, but the results it yields are far from being perfectly satisfactory, as the metallic sound so produced is not only not heard over the *whole* of the region corresponding to the stomach, but is also sometimes audible over the transverse colon when it is inflated with gas.—There is still another method, however, described by Frerichs,* whereby the outline of the stomach may be traced on the surface with somewhat greater precision. To the patient to be examined is administered a quantity of tartaric acid, followed up by a like quantity of bicarbonate of soda dissolved in water; effervescence takes place freely within the stomach, which is soon distended by the carbonic acid gas disengaged. When in this condition the stomach, which often forms a visibly prominent swelling in the epigastric region, gives a deep, tympanitic, and metallic percussion-sound, frequently of a strikingly different character from that rendered by the transverse colon.—Another method sometimes adopted to distinguish stomach from bowel by percussion, is to fill the former with fluid, when the sound obtained along its lower border, the patient being in the upright position, will be found to be dull.

The tympanitic gastric area which is here represented as associated with a medium state of dilatation of the stomach, may appear to be dislocated, increased, or decreased in size.

The stomach is *dislocated* downwards in those conditions in which the diaphragm is depressed,—copious pleuritic effusion, pulmonary emphysema; it is displaced upwards when the diaphragm is caused to rise to a higher level in the thorax, as by ascites, or large abdominal tumours.

The gastric resonant area is *diminished* by tumours of the liver and spleen, when these are sufficiently large to encroach upon the stomach to any extent.

Enlargement of the space which the stomach renders tympanitic to percussion occurs in cases of pathological dilatation of the organ. It is one of the signs frequently observed in gastric catarrh of long duration, and more especially in connection with stricture of the pylorus; in these circumstances the stomach is dilated along its greater curvature, its lower boundary sinks sometimes even as low as the umbilicus, while to the right it may extend far over the middle line, and to the left may pass beyond the axillary line, so that the whole upper, and occasionally also the middle portion of the abdominal cavity is occupied by the dilated organ. Throughout the whole of this region the percussion-sound is tympanitic, and at some parts may possibly be of a ringing metallic character, if the stomach contain chiefly air and only a relatively small proportion of fluid. If the stomach-contents consist mainly of fluid, and if the abdominal wall be soft and lax (as it generally is, these cases being usually marked by excessive emaciation), the gastric percussion-sound undergoes considerable modification on altering the position of the patient, and in this way the delimitation of the part may be greatly facilitated; thus, in the upright position the fluid gravitates to the lowest level within the stomach, and the sound along the greater curvature becomes much less resonant and more readily distinguishable from the clear tympanitic note of the colon. The lateral limits of the stomach may be determined in like manner, by turning the patient first to the right and then to the left side; the fluid, as before, seeks the lowest level, when the previously clear tympanitic percussion-sound at the points to which it sinks becomes feebler and duller.

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* In like manner Schreiber has proposed to pass into the stomach an œsophageal tube, to which is attached a small india-rubber balloon; this balloon, when it reaches the stomach, is inflated by blowing through the tube, and the organ is in this way distended with air.

The signs which the dilated stomach presents to inspection and palpation (see pp. 318 and 332) are often of the greatest value as aids to percussion in determining the limits of the affected organ; frequently, indeed, these are the only indications which can be trusted to in the diagnosis of the condition under discussion. In several cases of enormous dilatation which have come under my own notice the greater curvature of the stomach was recognisable as a well-marked, slightly prominent line or elevation which crossed the abdomen below the umbilicus, and which, on being smartly rubbed with the hand, showed peristaltic movements in relief with surprising distinctness. In two of these cases, in order to define the lower gastric boundary from the colon by means of percussion, I emptied the stomach with the stomach-pump and injected 4—5 kilogrammes of water; then only did the previously tympanitic sound become dull.

PERCUSSION OF THE INTESTINE.

The percussion-sound of the *great and small intestine* is, in health, always tympanitic. The pitch of this tympanitic sound is different at different points on the abdominal surface, and is subject to constant variation; this alteration in pitch is in general caused by the very variable condition of the bowel with regard to the amount of gaseous and solid matters it contains, and by the consequent variation in the tension of the walls of the intestine. Though a definite statement, therefore, of the points at which the tympanitic percussion-sound will be found to be lower or higher in pitch cannot be made, the general rule may nevertheless be laid down, that over the small intestine the sound is higher in pitch than over the large intestine.

The tympanitic intestinal sound is most exquisitely developed at those points at which the abdominal coverings are not too tense, at which, accordingly, they do not interfere with or muffle the percussion-note. It is thus always more or less diminished in resonance when the patient stands, this being the result chiefly of the tension of the recti muscles; a large accumulation of fat in the subcutaneous tissues has the same effect on the sound.

The raising and lowering of the pitch of the tympanitic intestinal sound so often observed in disease are generally referable to definite pathological causes. If there be an unduly small proportion of gaseous matters present in the intestine the latter is diminished in calibre, (as in ascites, in which the fluid presses on the bowel), and its percussion-sound rises in pitch; thus, in a case of ascites in which the small intestine is compressed into much less than its normal bulk, the sound above the level of the effusion is *acutely* tympanitic.—When, on the other hand, the bowel is tensely inflated with gas, the abdominal percussion-sound becomes deeper in pitch, but not tympanitic; this is observed, physiologically, after a heavy meal; pathologically it occurs in peritonitis, in typhoid fever (from paralysis of the muscular coats of the bowel), and in cases of stricture of the intestine, from the obstruction set up to the onward passage of the intestinal contents. The sound is lowered in pitch, because a larger body of air is thrown into vibration, but it is not rendered tympanitic, as the *wall* of the intestine as well as the *air* it surrounds is caused to vibrate (see p. 91).

If a portion of the bowel, situated above a stricture, be widely distended with gas, a ringing *metallic percussion-sound* is heard over it, while below the point of constriction the whole intestinal tube is reduced in lumen and therefore gives no such sound to percussion.

The intensity of the tympanitic note varies very much at different points on the surface of the abdomen, these changes being dependent on the amount and nature of the intestinal contents. The more solid the matters lying in the bowel, and the less the proportion of gas mixed up with them, the less the intensity of the tympanitic sound. Thus, at those parts beneath which lies the intestine loaded with large masses of fæcal material, the sound acquires the dull tympanitic character; in the left iliac region particularly, when the descending colon is filled with retained excreta, the percussion-note is less clear than at the corresponding point on the right side. Nevertheless the sound is never absolutely dull, even when the accumulation becomes enormous in amount.*

If, on the other hand, the intestine contain little solid matter but a large quantity of gas, the percussion-sound becomes very loud, but, at the same time, loses its tympanitic character if the intestinal walls are so tense as to be capable of entering into simultaneous vibration.

Accumulation of gas in the peritoneal sac, most commonly the result of perforation of the intestine, sometimes also owing to the disengagement of gas from a putrid peritoneal effusion, and in rarer cases caused by the bursting of a pyopneumothorax through the diaphragm, may give rise to the same intumescence of the abdomen as intestinal meteorism.—The percussion-sound in such cases is often in no way different from that of the last-mentioned affection; occasionally, however, it is of a ringing *metallic* quality. Further, if the gas burrow upwards between the anterior surface of the liver and the abdominal parietes, that is, to the highest point within the abdomen when the patient lies on his back, forcible percussion elicits a *clapping* or flapping sound, resembling that obtained over the stomach or bowel after removal from the body, due to the collision of the flaccid and only partially distended walls of these parts when struck with the percussing instrument. In both instances the sound originates in the sudden escape of the layer of air which is compressed by the percussion-stroke.† In cases of this nature, also, percussion of the lower and posterior parts of the abdomen usually reveals the presence of fluid effusion, as along with the intestinal gases a quantity of the liquid contents of the bowel escapes into the peritoneal cavity, and this very speedily excites a diffuse peritonitis with exudation, if the orifice in the intestinal wall be not at once closed by adhesive inflammation.

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* Barth records the case of a boy who presented two large intestinal fæcal tumours, one of which measured 12 ctm. in diameter, over which the percussion-sound was not absolutely dull, but of dull tympanitic quality. The mass which occupied the bowel was found, at the post mortem examination, to weigh 6 kilogrammes.

† Chomjakoff, on the contrary, explains the clapping sound heard on percussing

It is hardly possible to confound intestinal meteorism with an accumulation of gas in the peritoneal sac, in view of the violence of the symptoms by which the latter accident is attended,—the signs of perforation and subsequent peritonitis. The principal physical sign on which the diagnosis of the entrance of intestinal gases into the peritoneum is based is the disappearance of the hepatic dulness on both sides of the median line, as the bubbles of gas seldom fail to make their way upwards to the highest part of the cavity (see also the section on auscultation, p. 373).

FLUID EFFUSION IN THE PERITONEUM.

An effusion which does not rise above the pelvic cavity is not discoverable by percussion; as it increases gradually in quantity, however, and extends upwards into the abdomen, it renders dull the percussion-sound over more and more of the lower abdominal region when the patient stands erect. When the whole abdomen is filled with the fluid the sound is at all points dull, but the dulness becomes *absolute* only when the effusion is so abundant as to press heavily on the bowel, the latter being thus to a great extent emptied of air and so compressed as to occupy much less space than normally. Nevertheless, on sinking the pleximeter deeply into the abdominal tissues, and in this way pushing aside a portion of the fluid, the previously dull sound is made somewhat clearer by the accompanying tympanitic intestinal sound. Very frequently the air-filled coils of intestine are floated up on the surface of the fluid, and are therefore detected *above* the area of dulness; in such circumstances the sound will be found to be of clear tympanitic quality on percussing with a gentle stroke, and of the dull tympanitic variety on percussing forcibly and

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when bubbles of gas are interposed between the liver and the abdominal coverings as caused by the shock of these parts against each other when subjected to an energetic stroke. The explanation offered above, however, seems to me more natural and more in accord with that which is most generally recognised as satisfactory in the case of the bruit de pot fêlé,—a phenomenon to which the sign under discussion presents a close analogy.

pressing the pleximeter firmly down.—If the ascites be moderate in degree the dulness is always attended by the tympanitic intestinal sound.

The percussion-sound in ascites undergoes various *modifications* on *changing the attitude* of the body, as the fluid, in obedience to the laws of gravitation, subsides to the most dependent parts of the abdomen. On turning to the right side, accordingly, it sinks to the right, and to the left on turning to the left side, the sound invariably becoming dull at the deeper parts, and clear (tympanitic) in those which are for the time being uppermost and which are occupied by the movable mass of the intestines. Similarly the sound in the lower abdominal region, which in the upright position is rendered absolutely dull by the subsidence of the ascitic fluid to that part, clears considerably in the dorsal recumbent posture, as the effusion is then more equally diffused throughout the abdominal cavity. This modification of the percussion-sound with the varying attitude of the body is quite as positive an indication of the presence of free fluid in the abdomen as the sensation of fluctuation formerly described; it is a point therefore of high diagnostic importance, and one of which full advantage is always taken in the examination of cases of ascites. In doubtful cases, in which the effusion is limited to the deeper parts of the abdominal cavity, and is not abundant enough to yield the sensation of fluctuation, or to give rise to any very distinct muffling of the percussion-sound, a comparison of the sounds obtained in the flanks in the dorsal recumbent posture and in the right and left lateral positions is of itself generally sufficient to justify a positive diagnosis. It is only when the peritoneal effusion is unusually abundant that the percussion-sound of the uppermost side does not become quite clear, as the whole of the fluid does not then sink to the lowest part of the cavity. Nevertheless, even here the difference between the sounds is quite appreciable; it is obvious also that it is precisely in these extreme cases of ascites that this diagnostic aid is of least moment as a method of examination, the nature of the affection being evident enough from the other signs.

Percussion indicates also the level to which the effusion reaches in the abdominal cavity. Over the whole space taken up by the fluid, as far as its margin, the sound is dull; above this point, in the region corresponding to the situation of the intestine, the sound is clear, while the transition from dulness to perfect resonance is marked by a zone in which the percussion-note is muffled.

In cases in which the effusion is excessive in amount it is sometimes found to be impossible, when the patient is in the erect posture, to determine with absolute accuracy the upper margin of the body of fluid, though it may usually be defined to within ½—1 ctm. of its actual limit; this is owing to the muffling effect which the great tension of the upper part of the abdominal wall has on the percussion-sound. This degree of precision, however, is not demanded, as it is of no great practical value in deciding the question whether the fluid should or should not be discharged by operative puncture; the most weighty consideration in this respect is the amount of pressure the fluid exercises on the diaphragm, and consequently the degree of dyspnœa there is present, or the possible danger of suffocation. The operation is always performed at the most dependent part of the abdomen.

Ascites may occur alone or may be associated with general œdema of the subcutaneous cellular tissue. Ascites unconnected with any other form of fluid effusion is observed in those diseases of the abdominal organs which give rise to engorgement of the portal circulation, particularly in diseases of the liver (cirrhosis) and in the different varieties of degeneration of the peritoneum,—cancer, and more rarely tuberculosis.—Ascites is also found accompanying general dropsy, whether the latter be dependent on congestion of the systemic veins, as in cardiac and pulmonary affections, or on diseases which lead to impoverishment of the blood in respect of albumen, such as nephritis, amyloid degeneration of the kidneys, &c. In all these conditions ascites is observed only after œdema has appeared in other parts of the body,—in heart diseases, for example, usually only when the dropsical effusion has reached as high as the thigh.—Ascites is also often complicated by œdema of the abdominal wall.—The possibility of *mistaking ovarian dropsy for ascites*, which was referred to when treating of palpation (see p. 339), arises only when the wall of the ovarian cyst is enormously distended with fluid, becoming so thin that it cannot be felt through the abdominal tissues, the tumour then giving over its whole surface a uniform feeling of fluctuation. These affections are distinguished from each other partly by the results of vaginal examination, by which means the cyst may be reached with the finger, and partly by percussing while the patient is placed in the various positions already described; in ascites the *percussion sound varies* with every change of attitude on the part of the patient, in *ovarian dropsy* it *does not*. Occasionally, however, the two conditions are combined, the ascites arising either from some cause independent of the ovarian affection or from the pressure of the ovarian tumour on the portal vein. So long as the tumour is of moderate dimensions the existence side by side of both affections may almost always be demonstrated by palpation and percussion: the cyst may be defined by palpation, while the presence of the fluid around it is shown by the percussion-dulness to which it gives rise when the patient lies on her back, and by the change in the percussion-sound when she turns to one or other side.

In the event of the diagnosis being still doubtful even after careful physical examination, the manner in which the disease has developed, the presence or absence of morbid changes in the other organs, and many other incidental signs which may have a bearing chiefly on the particular case in hand, serve to distinguish clearly enough between ascites and ovarian dropsy.

ENCYSTED PERITONEAL EFFUSIONS.

These are formed by the adhesion of neighbouring parts to each other from inflammation of their peritoneal envelope, and the inclusion of the inflammatory exudation within the space so shut in. Peritoneal effusion may be encysted at any point within the abdomen; this occurs relatively most often in the ileocæcal region, where it is caused either by inflammation of the cæcum and of the peritoneum, or by perforation of the processus vermiformis.

The percussion-sound at the parts at which the fluid is confined is dull or muffled, the intensity of the dulness, other things being equal, being dependent on the amount of the exudation. The sound undergoes *no* alteration on changing the attitude of the body, a point which distinguishes it at once from free peritoneal effusion; fluctuation is wanting also, or is only feebly appreciable over a very circumscribed area.

The differential diagnosis between encysted exudation and tumours, local affections which may present substantially the same signs to palpation and percussion, is based on a consideration of the further details of the clinical examination, the history of the case, the origin and course of the disease, &c.

PERCUSSION OF THE KIDNEYS.

It is practically impossible to delimit the kidneys with any degree of precision by means of percussion. This fact is explained by the anatomical relations of the organs. In the first place, those parts of the posterior abdominal wall which coincide with the anatomical position of the kidneys, (the space included between the lower edge of the eleventh dorsal and the upper edge of the third lumbar vertebræ, according to Luschka), are covered by a thick layer of muscles, the sacrospinalis and quadratus lumborum, which of itself produces a very considerable muffling of the percussion-sound. Further, the kidneys in the greater part of their extent are in immediate relation with organs which give a dull sound to percussion: thus, the upper end of the right kidney is over-arched by the lower edge of the liver, the upper part of the left kidney borders on the spleen, the inner concave margins of both kidneys lie close to the transverse processes of the vertebræ,—all parts which are non-resonant to percussion. At only two points are the kidneys in contact with air-containing organs: they are in apposition with the colon along the lower half of their external convex border and at their lower ends, at which spot therefore the dull renal sound and the tympanitic note yielded by the colon may be sharply defined from each other. The capsule of fat in which the kidneys are embedded should also be mentioned as one of the factors which tend to increase the uncertainty which attends the delimitation of the organs by percussion. When this fatty envelope is well developed it may, according to the measurements of Pansch, be as thick below the kidneys as the kidneys themselves in their middle portion; in such cases therefore, which are far from being rare, the renal dulness extends further downwards than is in keeping with the position and size of the organs. In fact this dulness, notwithstanding that the lower end of the kidney comes generally only to within 3—5 ctm. of the crest of the ilium (Pansch), often reaches to very near this bone, where it is lost in the tympanitic note given by the colon.

From the foregoing account of the relations of the parts it will be obvious that at its upper and inner borders the kidney can not be delineated on the surface by means of percussion, that even at its lower end this can be accomplished only very imperfectly, and that the sole portion of the renal area that can be clearly marked off from the adjacent colon is its outer convex margin, from about the middle downwards, situated usually at 10 ctm. from the spinous processes of the corresponding lumbar vertebræ. At this point the dull renal sound is exchanged for the tympanitic note of the large intestine.

In very muscular subjects percussion of the kidneys yields absolutely no result which is of any practical diagnostic value; this applies also to the practice of percussion in those whose tissues are loaded with fat, or who suffer from extreme distension of the bowel, engorgement of the liver or spleen, ascites, &c. Bearing in mind the many difficulties with which the examination of the kidneys by percussion is surrounded, and the actual impossibility of determining the size of the organs with anything approaching precision, it will be readily understood why percussion should be so seldom resorted to as a means of diagnosis in renal diseases, and that its applicability should be limited almost exclusively to the rather rare cases of dislocated (movable, floating) kidney. In this condition the lumbar region on the side affected is sometimes clearer to percussion than the other; in two out of six cases of dislocation of the right kidney which I have observed, the sound over a somewhat extensive area in the right lumbar region was of dull tympanitic quality, while in the four remaining cases no difference whatever was discernible between the two sides. In none of the other diseases of the kidneys is percussion employed as a method of physical examination, as the indications it furnishes are not reliable. The most common of the diseases of the kidneys, the various forms of nephritis, are recognised with so much ease and certainty by the changes to which they give rise in the urine and by the occurrence of dropsy, that as a rule there is no necessity for falling back on percussion, and there is the less occasion for it as the kidney, when inflamed, undergoes no such degree of enlargement as is discoverable in this way. It is likewise impossible to demonstrate by percussion the contraction of the kidney which takes place in the third stage of nephritis. There remain, therefore, as objects to which percussion of the kidneys may be advantageously directed, only the larger kinds of renal tumours, such as those due to hydronephrosis. The sound in such cases is dull over a very wide area; even here, however, percussion is not indispensable, as the swelling may be distinctly felt with the hand through the abdominal wall.

PERCUSSION OF THE BLADDER.

The bladder, when empty, does not rise above the pubes; when widely distended with urine, however, it projects upwards from the pelvis into the abdomen and forms in the hypogastric region, provided the skin of the part be lax and soft, a pyriform elastic tumour which is readily detected by palpation.—The percussion-sound over a bladder tensely distended with urine is absolutely dull. All risk of confounding such a swelling with any other pathological condition is obviated by simply introducing a catheter into the bladder; if on account of the state of the parts (as from enormous hypertrophy of the prostate) it is found to be impossible to pass an instrument, the question whether the tumour is or is not the bladder,—a question of prime importance, having in view the fact that in such cases puncture of the bladder is sometimes called for—may be settled by a consideration of the history of the case, by the circumstance that for some time no urine, or but a very small quantity, has been passed, and by noting that the swelling, in contradistinction to that of ascites, retains the same dull percussion-sound in all the different attitudes that the patient assumes. The non-resonance to percussion distinguishes this condition at once from intestinal meteorism. It occasionally happens, though comparatively rarely, that a coil of intestine becomes fixed between the bladder and the abdominal wall; here the sound, on percussing gently, is of a tolerably clear tympanitic character, that obtained in answer to a more forcible stroke being of a dull tympanitic quality. The degree of distension of the bladder, if the upper end of the organ be not appreciable by inspection and palpation, is indicated by the upper boundary of the area of dulness.

PERCUSSION OF THE UTERUS.

During pregnancy, or in cases of pathological enlargement, percussion of the uterus gives no information which cannot be elicited with much greater distinctness by palpation. The percussion-sound over the whole of the enlarged organ is absolutely dull. The womb, however, becomes accessible to percussion only when it has already become equally accessible to palpation, that is, when it rises out of the pelvis into the abdomen.

As a general rule the enlarged uterus lies immediately behind the abdominal wall, though in certain cases these parts are separated by coils of intestine, when the percussion-sound takes on the same modifications as that of the bladder under similar circumstances.

The uterus after it has emerged from the pelvic cavity, and the bladder when it is fully distended with urine, both give a dull sound to percussion, and may possibly therefore, though this is highly improbable, be mistaken for each other; but the diagnosis presents no real difficulty as the vesical swelling rapidly disappears on passing a catheter and drawing off the retained urine, while the enlarged uterus gives to palpation a feeling of increased resistance which is quite distinctive.

AUSCULTATION OF THE ABDOMINAL ORGANS.

The phenomena observed on auscultating the abdominal organs pertain, with the exception of the sound heard over the uterus in the later stages of pregnancy, almost exclusively to the digestive apparatus; they are constant in only a few isolated pathological conditions, and are as a rule merely accidental and irregular in occurrence, though at times they may also be produced at will. A *methodical* application of auscultation in the examination of the abdominal organs is thus scarcely practicable; it is employed as a means of diagnosis only occasionally, therefore, when dealing with affections which are known to present signs appreciable by auscultation. It is of importance, however, to be acquainted with the various sounds which originate in the gastro-intestinal canal, as certain of them reach the ear when auscultating the lower part of the thorax and the cardiac region, where they sometimes accompany and modify the respiratory murmur and the heart-sounds and murmurs in such a way as to prove perplexing to the inexperienced examiner, who is apt to consider them as sounds actually developed within the chest.

Auscultation of the Œsophagus.

This method of examination, recommended strongly by Hamburger as an aid to diagnosis in diseases of the œsophagus, is based on the circumstance that the act of deglutition, whether the substances swallowed be fluid or solid, is attended by the production of certain characteristic sounds in the upper part of the alimentary tract.

The cervical part of the œsophagus is best auscultated by placing the stethoscope on the left side of the neck, close to and behind the trachea, at any point between the hyoid bone and the supraclavicular fossa; murmurs arising in the thoracic portion are most readily recognised close to the left side of the spine, from the level of the last cervical to the eighth dorsal vertebra.

In normal conditions there is heard in the cervical portion of the œsophagus a ringing gurgling sound when fluid is swallowed; a similar sound, but weaker, is audible over the thoracic part. In *stenosis* of the œsophagus, when the constriction is so great that even fluid passes slowly and with difficulty, it is sometimes noticed that below the stricture the sound follows deglutition at an abnormally long interval and is also to some extent enfeebled, but these signs never occur with such regularity or constancy as to enable one to determine the precise point at which the canal is narrowed. The fact that in such cases fluid takes a longer time to reach the stomach and enters it more slowly is also much more clearly appreciable on auscultating in the epigastrium than over the thoracic part of the œsophagus. In a case of inversion of the viscera which I examined the situation of the œsophagus to the right was indicated by the presence of deglutition-murmurs on that side of the neck and their absence on the left. A much more marked murmur, and one therefore much better adapted for auscultation, is generated by drinking fluids than by swallowing solids.

Auscultation of the Gastro-Intestinal Canal.

The phenomena revealed by auscultation of the gastro-intestinal canal consist of murmurs, of which some are spontaneous while others (and the greater number) are produced voluntarily. The presence of fluid in the stomach or bowel is necessary to the existence of these murmurs; they are in a few rare cases due to other causes, which will be mentioned further on. On agitating the fluid contained in the stomach or intestine by pressing repeatedly and quickly on the abdomen, a loud ringing metallic splashing or gurgling sound is heard, which may be detected without bringing the ear close to the abdominal wall, or even at some distance from the patient. It is identical with the Hippocratic succussion-sound observed in pneumothorax, or the sound caused by shaking up fluid in a jug, and is ascribable to the same physical conditions,—the agitation of fluid and air in a closed cavity presenting the conformation, &c., necessary to the development of *consonance*.

These murmurs, particularly those arising in the stomach, are very frequently noticed, in perfectly healthy persons shortly after food or a large volume of fluid has been taken, when the contents of the stomach are thrown into commotion by making sudden pressure on the upper part of the abdomen. They also sometimes originate spontaneously, independently of any mechanical disturbance of the parts adjoining the stomach, but are then scanty and feeble and audible only through the stethoscope or with the ear applied directly to the surface. They not unfrequently seem to depend on the upward and downward movement communicated to the stomach during respiration. Even the simple movement of the parts concerned in the act of deglutition may be accompanied by a scanty murmur, appreciable on auscultating the stomach, and closely resembling the ringing metallic ráles; on actually swallowing a little fluid, moreover, these sounds increase greatly in intensity.

The spontaneous gastric murmurs above mentioned are sometimes so distinctly audible over the posterior lower part of the thorax and in the cardiac region that one unskilled in auscultation, when examining these parts, might very readily fall into the error of regarding them as true pulmonary metallic râles; their generally rapid disappearance, however, their irregularity with respect to the period of respiration at which they occur, and the fact that they are heard even when respiration is suspended, are all points which suffice to stamp them as murmurs arising in the stomach or in the adjacent portion of the colon.

When the *stomach*, dilated from pathological causes, contains a large quantity of fluid as well as gas, and when in addition to this its walls are soft and flaccid (as they generally are), sudden pressure in the upper abdominal region gives rise to numerous ringing splashing or gurgling sounds, frequently audible even at a distance,—sounds which, in proportion to the violence of the disturbance of the stomach, are distributed over a large part of the abdominal surface superiorly or even, as I observed in one case, over nearly the whole of the abdomen. This physical sign, together with others already mentioned, is diagnostic of dilatation of the stomach or of that condition of the organ in which food and fluid are detained in it for an unusually long period. In such cases also a number of fine râles may be detected on auscultating, without making any pressure with the hand on or near the epigastrium; these spontaneous râles are doubtless due to fermentation of the contents of the stomach and the consequent formation and bursting of bubbles of gas.

Gurgling or splashing sounds similar to those arising within the stomach may be produced also in the *intestine,* when its contents are thin and watery, by shaking and pressing upon the abdomen. This is observed in *all* cases of *diarrhœa;* it is the louder the greater the quantity of fluid, and is therefore exceedingly well marked in cholera, in which the paralysed intestine is often found enormously distended with watery discharge. This gurgling may be audible over the whole abdomen—though not usually in equal intensity at all points—or only at certain parts. Of those coming under the latter category, the circumscribed intestinal sounds, the most note-worthy from the diagnostic point of view is that elicited by pressure and limited to the *ileo-cæcal* region, occurring especially in typhoid fever, but met with also in simple catarrh of the ileum and cæcum.—Gurgling may also be developed spontaneously within the bowel in cases of diarrhœa, when the fluids are urged quickly onward from one part of the intestinal tube to another by powerful peristaltic action or when the bowel is compressed by the contraction of the abdominal muscles; in these circumstances the action of the muscles of the abdomen and intestine gives rise to the same phenomena which in the cases mentioned above are mechanically excited by the force of the hand of the examiner.

Such sounds, however, do not depend solely on the presence of fluid in the intestinal canal; they may be caused by the rapid passage of *gas* through the bowel, as the result of increased peristaltic action.

With these rumbling noises (*borborygmi*), in Germany described onomatopoietically by the terms "kollern, poltern, gurren," every one is sufficiently familiar; they are often heard when the stomach and bowel are empty, as after a prolonged fast, very frequently in diarrhœal conditions they are the immediate precursors of an evacuation of the bowels, while in many who are subject to flatulence their continual occurrence becomes a troublesome and annoying habit. In diarrhœa they are sometimes accompanied by colicky pains passing rapidly over the abdomen in the direction of the colon. Not unfrequently they cease spontaneously, but relief is complete and lasting only when the wind escapes by the anus.

In cases of *perforation of the bowel, murmurs* of a blowing *amphoric* character may be developed, when the opening is sufficiently wide to permit of the free intercommunication of the gases contained in the intestine with those which have already entered the peritoneal sac. These sounds are synchronous with the respiratory movements, and are louder in inspiration than in expiration (Tschudnowsky); it is to the inspiratory descent and the expiratory ascent of the diaphragm that they are to be ascribed, as by the former the intra-abdominal pressure is greatly increased and a quantity of gas driven through the opening into the peritoneal cavity, while by the latter the pressure is relieved and a certain proportion of the gas caused to re-enter the bowel through the perforation. This amphoric sound, on the other hand, when it is not spontaneously audible and does not happen to coincide with the respiratory movements, may be artificially produced by exercising sudden rapid pressure on the abdomen and at the same time listening either with the ear applied directly to the surface or through the stethoscope; the gas is displaced from the parts under the hand, a portion of it passing into the intestine when the perforation is still patent and of sufficient width (Sommerbrodt). If, however, the opening in the bowel be small, as it most commonly is, or closed up by inflammatory products, no trace of these amphoric sounds can be discovered; but as in peritonitis from perforation of the intestine the cavity contains not only air but a variable amount of fluid, a *ringing metallic splashing sound* (abdominal succussion) is heard on pressing firmly and quickly on the abdomen, or on grasping it between the hands and shaking it,—provided such rough handling is bearable, considering the acuteness of the pain by which these cases are usually characterised. The severity of the symptoms which accompany peritonitis from this cause affords a ready means of distinguishing these sounds from the splashing or gurgling developed in the stomach or intestinal canal. The sounds of the abdominal aorta also acquire a metallic quality when the peritoneal sac is filled with gas.—Sometimes, though rarely, the evolution of gas, taking place within closed abdominal cavities containing pus, (encysted exudations, cysts), gives rise to similar splashing sounds (Laboulbène, Gerhardt, &c.).

The *peritoneal friction-murmurs* which are met with in certain cases are palpable as well as audible, and have already been discussed in the chapter on palpation (see p. 336).—It should be mentioned also that occasionally, on pressing on the gall-bladder when it is occupied by a number of large calculi, the *clinking* of these against each other may be felt and heard.

Vascular murmurs are also sometimes observed in cases of abdominal *tumour*. They are most commonly associated with tumour of the uterus (occurring, according to Spencer Wells and Winckel, in about one-half the cases), more seldom with ovarian tumour; Winckel and Hirschfeld report having detected them in subjects suffering from tumour of the spleen, and Leopold has heard them once in hepatic cancer. Their arterial origin is shown by their being synchronous with the pulse.

Auscultation of the Gravid Uterus.

The aim of auscultation of the gravid uterus is the detection of the sounds of the fœtal heart and the placental murmur.

The *sounds of the fœtal heart*, discovered by Mayor, of Geneva, in 1818, are first appreciable towards the end of the fourth month of pregnancy. At this period they are feeble, but subsequently they gain considerably in volume; they are most often heard on the left side of the mother, from the great frequency of the first cranial presentation, in which the back of the child is turned forwards and to the left. Their presence proves conclusively that the child is alive, their absence that it is dead, auscultation thus furnishing an important indication for the adoption or the avoidance of operative interference with the process of delivery.

The *placental murmur*, discovered by Lejumeau de Kergaradec, in 1822, is a sound which is synchronous with the arterial pulse, but is frequently of slightly longer duration, particularly when the abdomen is somewhat forcibly pressed upon with the hand. It is not of equal intensity at all points on the surface of the womb, being louder sometimes to the right side, sometimes to the left. It is heard in the second half of pregnancy, being at first very faint, but afterwards of greater intensity. It is developed in the dilated uterine arteries, at the part at which they terminate in the uterine veins.

EXAMINATION OF THE EXCRETA.

THE URINE.

THE points to be noted in examining the urine are its quantity, colour, reaction, specific gravity, and the occurrence in it of abnormal constituents.

QUANTITY OF THE URINE.

This is very variable even in perfect health, and depends chiefly on the amount of fluid taken by the patient and on the greater or less abundance of the excretion of water by the sudoriparous glands of the skin. If the consumption of fluid be moderate and the cutaneous transpiration slight, as in winter, the quantity of urine passed by an adult in the twenty-four hours amounts on the average to about 1,500 ccm.

The urine is *diminished* in quantity in all febrile affections, in the stage of failure of compensation in diseases of the heart, and frequently in the different forms of *nephritis* (though in the latter class of cases it sometimes remains normal in volume); it is, further, reduced in a number of other disorders, and occasionally even in health, from causes of only temporary duration, to be mentioned in detail further on.—The quantity voided in the twenty-four hours may, in extreme cases, sink to one-fourth that passed normally; at times even, as in the stage of collapse in cholera, and now and then also in scarlatina, almost complete anuria may be observed.—The cause of this diminished excretion is either that the quantity of blood in circulation is unusually small or that the blood itself is deficient in water, the pressure within the renal arteries being in both cases reduced to a minimum. When in the later stages of cardiac disorders the compensatory changes in the heart's structure fail to completely overcome the obstacle to circulation, the venous system becomes overloaded, the arteries, and among them those of the kidneys, contain less blood than normally, and the excretion of urine naturally goes on but slowly; the urine is scanty also in other conditions in which the blood is not sufficiently fluid, either from the escape of its watery constituents through the walls of the veins, from increase of the cutaneous transpiration, or frequently as the result of the undue abstraction of water from the system by the very abundant and thin discharge from the bowel in diarrhœa and the allied affections. Diminution in the amount of urine excreted, with whatever form of disease it may be associated, and whether it be only a passing complication or a phenomenon of a more persistent character, may generally be easily accounted for by a careful consideration of the series of causes just enumerated.

The flow of urine is greatly *augmented* in *diabetes* mellitus and diabetes insipidus, the intense thirst from which the patients suffer causing them to drink very freely of water; the amount passed daily may increase to three, four, or even eight times that of a healthy person.

A moderate increase in the quantity of the urine may often be produced by stimulating the kidneys by the employment of diuretics; diuresis also appears when, from any cause, the arterial pressure is raised.

Colour of the Urine.

Normal urine varies in colour from pale yellow to clear amber yellow or yellowish-red, the precise shade being determined by the greater or less abundance of the urinary pigments or by the degree of concentration of the urine. It has been calculated that in a healthy urine of clear amber colour there are eight parts of colouring matter to every thousand of fluid, and in a yellowish-red urine sixteen parts to the thousand (Vogel).

Normal urine contains at least two pigments, possibly more. The best known of these is urobilin, first separated by Jaffe; it resembles the colouring matter of the bile (bilirubin), is allied to hæmatin, and is found in the contents of the intestine and in fæcal masses, which owe to it their brown coloration (Vanlair and Masius, Maly). Hoppe-Seyler affirms, however, that urobilin is not one of the actual constituents of the urine, but that the colour of the excretion is due to only one substance, from which, after it has been precipitated by acetate of lead and again separated from the lead by sulphuric acid and alcohol, urobilin is formed by gradual spontaneous oxidation.—Another pigment, *uroerythrin* (or purpurin) gives to the sediment of uric acid and the urates its yellowish-red or brick-red colour.

The urine becomes *reddish yellow or red* in *all febrile diseases,* chiefly from the increased formation of the red colouring matter but partly also from the scantiness and consequent concentration of the urine in fever. This reddish tint is also observed, of various degrees of intensity, in chronic affections, when the volume of urine excreted diminishes to any very marked extent, particularly in the stage of failure of compensation in cardiac disorders.—The urine may, on the contrary, be abnormally *pale* from absolute deficiency of pigment; this occurs frequently in convalescence from severe acute diseases, in anæmic chlorotic conditions, and as the result chiefly of dilution in both forms of diabetes.

Of the abnormal colouring matters which may be present in the urine the most common are those of the biliary secretion and the blood.

A large proportion of *biliary pigment* (bilirubin) gives to the urine a brownish colour, with an occasional admixture of yellowish green or brownish green; an abundant foam, the yellow or green hue of which is more evident than that of the rest of the urine, forms on its surface when shaken. Strips of linen or blotting-paper dipped in such a urine at once take on a marked yellow coloration.—The most convenient test for biliary pigment is that proposed by Gmelin, in which the extremely delicate reaction of bilirubin on impure nitric acid (that containing nitrous acid) is made use of, the test fluid being prepared by adding to a small quantity of the chemically pure nitric acid a few drops of the fuming acid. The icteric urine is poured slowly, drop by drop, down the inside of the test-tube at the bottom of which lies a little of this impure acid, when at the point at which the fluids meet will appear a series of beautiful rings of colour arranged in the following order: the upper ring is bright grass-green, the next below it blue, the others in succession violet, red, and yellow.

Usually only the green, violet, and red rings are constant and retain their colour for any length of time, the blue and yellow being often indistinguishably mixed up with the other shades. The green ring is the only really characteristic indication of the presence of biliary pigment, as the other colours, especially the reddish-violet tints, appear also in urine containing no bile, on the addition of strong nitric acid, the change, in the latter case, showing that another pigment, indican, is present. The more slowly and carefully the urine is added to the nitric acid in the tube the broader will be the upper green ring; the green coloration is also the more intense the more bilirubin there is in the urine.—On allowing a drop of nitric acid to fall on blotting-paper previously stained with the icteric urine a similar green-coloured ring is obtained.

If the biliary pigment be present only in traces it may be separated and recognised by shaking up a quantity of the urine with chloroform; the latter dissolves out the bilirubin, acquiring in this way a yellowish colour; being specifically heavier than the urine, also, it falls to the bottom of the glass.—Ultzmann has recently stated that in cases in which the other ordinary tests fail, bilirubin may be detected in the urine in the following simple way: 10 ccm. of the suspected urine are mixed with 3—4 ccm. of pure concentrated potash lye (1 : 3 of water), and neutralized and finally acidified by adding to it pure hydrochloric acid; as the fluid becomes acid a beautiful emerald-green coloration is developed.—A very striking green coloration is produced on the addition of a few drops of tincture of iodine to the icteric urine (W. Smith).

Bilirubin is found in the urine in all affections attended by jaundice; it may disappear from the urine, however, while the icterus still persists.—Traces of the biliary acids also usually accompany the biliary pigment in the urine.

Blood communicates to the urine a decided red colour, the depth of which depends on the proportion of the abnormal element present. If the blood has been diffused through the urine for some time, and its colouring matter in this way altered, the urine may be reddish brown, brownish black, or even inky black. The colour presented by the urine, even when the blood-pigment has undergone change, and also when the quantity of blood contained is but small, is usually so characteristic that it is almost impossible to mistake it for any other variety of abnormal coloration (such as that caused by the increase of the normal urinary pigment observed in fever, &c.)

The colouring matter of the blood (oxyhæmoglobin) is always liberated from the corpuscles and dissolved by the fluid basis of the urine; it is only in those instances in which the proportion of blood present is exceedingly large, in which accordingly the process of diffusion between the urine and the blood globules is practically arrested, that the red corpuscles still retain their pigment. When urine containing blood is allowed to stand some time, the corpuscles subside to the bottom of the vessel as a red sediment.—Microscopic examination shows the blood disks either distended and more or less pale in colour, or shrunken and crumpled and of the irregularly dentate outline which they assume in saline solutions. Some cases are met with, however, in which, notwithstanding that the urine is of a deep *blood red colour, the blood corpuscles are completely wanting*, even in urine freshly passed. This variety of the affection is designated *hæmoglobinuria*. It indicates that the disintegration of the blood globules has taken place within the organism itself, the liberated colouring matter then passing directly into the urine. The causes which bring about this destruction of the corpuscles, and in this way produce temporary or persistent hæmoglobinuria, are still obscure.*

In those cases in which the urine has a reddish blood colour but shows no trace of corpuscles on microscopic examination, the presence of the blood pigment may be readily demonstrated by certain *chemical reactions*, and more clearly still by *spectrum analysis*. Of the various chemical reactions given by the colouring substance of the blood probably the easiest to obtain is that observed on boiling the urine after adding to it a little caustic soda or potash; brownish red coagula are formed, consisting of the precipitated phosphates and hæmatin (Heller). The slightest trace of oxyhæmoglobin in a fluid, even as little as 1 part in 10,000, is recognised in the *spectroscope* by the appearance of two separate absorption-bands between Fraunhofer's lines D and E in the *yellow* and *green* of the spectrum. After urine containing blood has stood for some time the oxyhæmoglobin is gradually transformed into *methæmoglobin;* this is simply one step in the series of changes through which oxyhæmoglobin passes in breaking up into hæmatin and albuminous matters. Such a urine is reddish brown in colour, and shows in the spectrum only *one* absorption-band, between the lines C and D. In those urines also which contain blood but from which the red corpuscles have disappeared the oxyhæmoglobin is converted into methæmoglobin.

The blood which is mixed with the urine may be derived from any part of the urinary tract; at one time it is observed only in traces, at another the urine seems to consist almost exclusively of a perfectly sanguinolent fluid.

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* Hæmoglobinuria has been observed in scurvy, septic fevers, and sulphuric acid poisoning, occasionally as one of the effects of the inhalation of arseniuretted hydrogen (Vogel), and in a case resembling intermittent fever (Secchi). In animals (rabbits) the subcutaneous injection of glycerine is followed by a red sanguineous staining of the urine (Luchsinger), which is caused, as I found, not by the presence of the red corpuscles themselves, but simply by the colouring matter of the blood.

The quantity of blood discharged is by no means a sure guide to the part from which it comes, this being indicated rather by the symptoms and appearances as a whole and by the result of microscopic examination of the urine; thus, the hæmaturia is obviously not due to kidney affection if on repeated examination no casts or epithelial scales from the tubuli uriniferi are found.

Several other urinary pigments have been observed, all of them resulting from the decomposition of certain of the normal constituents of the urine. Of these the most important is *indican*. Indican occurs in small quantities in every urine; in decomposing urine it is often seen as a bluish red glittering appearance or as a blue pellicle floating on the surface. The best method of demonstrating the existence of indican in the urine is that devised by Jaffe: to the urine is added an equal volume of hydrochloric acid, and then a few drops of a strong solution of chloride of lime, when the indican is decomposed, indigo is formed, and the urine takes on a *blue* coloration, which is the deeper the larger the proportion of indican present. If the indican be very abundant, a flocculent precipitate of indigo-blue (uroglaucin) is thrown down. Heller's test for indican,—mixing equal volumes of urine and fuming hydrochloric acid, and heating the mixture over a lamp,—gives a beautiful reddish violet coloration (indigo-red, urrhodin), the intensity of which varies with the amount of indican in the urine so treated; if this be unusually large the indigo is separated by this method also as a blue precipitate.

Increase of the indican takes place very frequently in the most diverse diseased conditions, particularly in *consumptive* affections and all disorders accompanied by much debility (Senator); to this category belong diseases of the stomach and bowels, especially cancer of the stomach, ileus, peritonitis, phthisis, &c. The quantity of urinary indigo may rise in ileus and diffuse peritonitis to 50—100, or even 150 milligrammes per day, the normal amount being only 5—20 milligrammes daily (Jaffe).

Brown or *inky black* pigments sometimes appear in the urine of patients suffering from *melanotic cancer*. When recently voided such urine is generally still free of any brownish discoloration; this is developed only after the urine has for some time been in contact with the air, or on adding to it oxidising substances such as chlorate of potash, chromic acid, nitric acid, &c. It is not yet certain whether this brown or black colouring matter is really of pathological origin or is merely one of the normal urinary pigments present in unduly great quantity; it is most probably the former, however, as it differs from the other familiar colouring substances found in the urine in offering greater resistance to their usual solvents (Ganghofner).

There is still another brown pigment sometimes discovered in the urine, and due to the presence of *catechin;* it is formed only after the urine has stood for a considerable period in contact with the air or when decomposition is beginning, and has hitherto been observed in only a few cases in the human subject (Müller and Ebstein, Rajewsky, Baumann). A urine containing this substance becomes intensely green on adding to it one drop of chloride of iron, and this colour changes to violet on the further addition of ammonia or the bicarbonate of soda.

The urine may also incidentally and temporarily be abnormal in colour after the internal administration of certain medicines; after rhubarb and senna it becomes brownish red (from the formation of chrysophanic acid), almost black after inunction with tar and particularly after the internal use of carbolic acid, and yellow, like the urine in jaundice, after santonin.

REACTION OF THE URINE.

This is usually *acid;* blue litmus paper dipped in the urine is coloured red, while red litmus paper remains unaffected. The acid reaction is owing chiefly to the *acid phosphate of soda* which the urine contains, though sometimes it may be partly due also to free uric acid and acid urates.

The degree of acidity is even in normal conditions very variable, and is estimated according to the intensity of the reddening of the blue litmus paper. Urine of very acid reaction is often associated with inflammatory febrile diseases, especially with acute articular rheumatism, &c.

The urine becomes *alkaline* after exposure to the air for some days. The occurrence of this change, which has been designated alkaline fermentation, is favoured by a high temperature; it is caused by the decomposition of the urea of the urine and its conversion into carbonate of ammonia, probably under the influence of the fungus-germs and bacteria which are suspended in the air and which drop thence into the urine. Alkaline urine renders red litmus paper blue, has a powerful smell of ammonia, and gives a white vapour of chloride of ammonium on holding over it a glass rod dipped in hydrochloric acid.

Even directly after emission the urine may, in a variety of circumstances, give for a longer or shorter period an alkaline or neutral reaction; in the latter case it does not change the colou of either red or blue litmus paper. The urinary secretion is alkalised by the prolonged internal administration of carbonate of soda or potash (in alkaline mineral waters), or of the alkaline salts of the vegetable acids (in various kinds of fruits), all of which appear in the urine as alkaline carbonates. The reaction of the urine is found also to depend to some extent on the secretion of the acid gastric juice; if the acids needed for digestion be (in animals) neutralized by the exhibition of carbonate of lime or magnesia (Maly), or if they be removed from the stomach by washing (Quincke), the urine speedily becomes neutral or even alkaline. The fact also that the acidity of the gastric juice is expended on the food accounts for the neutral or feebly alkaline reaction of the urine so often noticed for a few hours after a meal. In diseases of the stomach, in which from many causes (as from frequent vomiting) very little of the gastric acid is retained, the urine is not uncommonly distinctly alkaline. It is still more markedly alkaline when loaded with pus,—in catarrh of the bladder, in pyelitis, &c.,—when it may, even when recently voided, have a strong ammoniacal odour; if the quantity of pus be moderate, however, the urine does not usually become alkaline, but much more feebly acid than in health. Alkaline fermentation of the urine may sometimes be set up within the bladder by the introduction of a catheter which has not been previously made scrupulously clean. Urine which is alkaline when passed is invariably opaque and turbid, from precipitation of the phosphates; recent urine, when alkaline, may even contain crystals of ammoniaco-magnesian phosphate, exactly as in the case of a urine which has undergone alkaline fermentation outside the body.

Specific Gravity of the Urine.

The specific gravity of the urine is estimated by means of the aræometer (the urinometer). This instrument is placed in a cylindrical vessel filled with the urine to be examined, in which it should swim freely; it sinks the deeper the lighter the urine, and *vice versâ*. The density is indicated by the number on the graduated scale which, when the instrument comes to a state of rest, is found to be on a level with the upper surface of the fluid. Normally the specific gravity of the urine varies from 1015—1020, that of distilled water being regarded as 1000.

In *disease* the density of the urine may mount to 1040 or sink as low as 1005; in many cases even these limits are considerably overstepped.

The specific gravity of the urine *rises*, to 1025 or even a slightly higher point, in *febrile* diseases. This is the result chiefly of *increase of the excretion of urea;* it seems also to be owing partly, but to only a very limited extent, to increase of the other solid urinary constituents (the urates). Some of the solid elements of the urine may even be diminished in febrile conditions; thus, in inflammatory exudative diseases, especially in *pneumonia*, the *chlorides* may almost or entirely disappear from the urine (Redtenbacher). The degree of dilution of the urine also takes an important place among the factors which determine its specific gravity. If the same weight of solid matters be at one time dissolved in a large volume, and at another in a small volume of water, the density of the fluid in the former case falls, and in the latter case rises. The amount of water in the urine is reduced in fever, and in all those conditions which are followed by dropsy and ascites,—in the stage of failure of compensation in heart diseases, in affections of the liver, &c.; in these disorders therefore the density of the urine is increased, a specific gravity of 1030 being generally reached, but not commonly exceeded. Urines which, from deficiency of water, the absolute quantity of solid constituents being either unaltered or possibly increased, possess a density so high, are invariably dark in colour.

But there is another condition in which, notwithstanding that the watery portion of the secretion is greatly augmented, the density of the urine *rises*, from the presence of *sugar* along with the normal urinary solids. Such a urine is distinguished from those just described by its perfect clearness and its pale yellowish colour. The lowest density of urine containing grape sugar (diabetes mellitus) is about 1030; usually it ranges from 1032—1040, seldom rising above 1040—1050, and only in the very rarest cases reaching 1060—1065 (Seegen). The specific gravity, however, is not exactly proportionate to the amount of sugar present, being influenced also by the other solid constituents of the urine, whose quantity may be simultaneously increased or diminished. The proportion of sugar contained in the urine cannot thus be inferred with absolute certainty from its specific gravity.

The density of the urine is sometimes *lowered* temporarily in perfect health, after a large draught of water, the total quantity of urine secreted being in proportion to the amount of fluid taken; the density may from this cause rapidly fall even to 1005. If, however, the specific gravity sinks, notwithstanding that the urinary secretion is *not* augmented, we have before us a fact of distinctly pathological signification. This is observed in anæmic conditions, and frequently also in nephritis; the lowering of the density is here due principally to diminished separation of urea by the kidneys.—Decrease in the density of the urine occurs also in *diabetes insipidus*, from pathological increase of the watery part of the urine, independently altogether of any alteration in the excretion of urea or of the other solid constituents.—All urines of low specific gravity are pale.

Trapp's formula furnishes a ready method of estimating approximately the solids held in solution in the urine. If the two last figures of the specific gravity be multiplied by 2·3, the product indicates in grammes the amount of solid matters present in 1000 ccm. of the urine; thus, if the density be 1015, 1000 ccm. of such a urine will contain 34·5 grammes of solids.

The solid constituents of the urine consist of about equal parts of *inorganic salts* and *urea*. Of the former, the *chloride of sodium* occupies the first place, being excreted to the extent of 10—15 grm. per day; the *sulphates* amount on the average to 2 grm., the *earthy phosphates* to 1 grm., in the 24 hours, while the *salts of ammonia* are detected only in traces.—Of the *organic urinary solids* the most important is *urea*. It constitutes 2½—3 per cent. of the urinary secretion; the other organic elements are much less abundant, ½—⅓ grm. of *kreatinine*, 0·2—1 grm. of *uric acid*, ⅓—½ grm. of *hippuric acid*, being discharged in the twenty-four hours. The urinary pigments have already been discussed.—Several other organic substances (xanthine, oxalates, &c.,) occasionally appear in the urine, but only in minute traces.

ABNORMAL SUBSTANCES IN THE URINE.

Albumen. This substance passes from the blood into the urine when the blood pressure within the renal veins is increased, whether from inflammation of the kidneys (which is the commonest of all the causes of albuminuria, and gives rise to the phenomenon in its most fully developed form) or as the result of diseases of the circulatory, and sometimes also of the respiratory apparatus, when they produce engorgement of all the systemic veins, and, among them, of the renal veins; or the albumen is one of the signs of the presence of extravasated blood or of pus, which may be mingled with the urine at any part of the uropoietic system; or, finally, it may proceed from other secretions which may have gained admission to the urine accidentally.

The presence of albumen in urine is demonstrated by precipitation. In urine of acid reaction the albumen is thrown down on heating to the boiling-point, by the addition of nitric acid, &c. To ensure accuracy it is better to employ both methods, heating and the use of nitric acid, in examining for albumen, as the phosphate of lime falls as a white precipitate on simply warming the urine, but is again dissolved on adding a few drops of the acid, when the urine becomes clear; *albumen* which has been precipitated by boiling, on the contrary, is unaffected by the nitric acid, or if the first part of the process has been insufficient to coagulate the whole of it the acid completes the precipitation. On the other hand, neither is the simple addition of nitric acid to the cold urine a test which is absolutely free from fallacy, as it produces a slight degree of turbidity in the presence of urates; this cloud is cleared away, however, if the urine be non-albuminous, on applying heat, as the amorphous urates are in this way at once dissolved. When the precipitate is abundant it is always easy to decide, even by simple inspection, whether it consists of albumen, phosphates, or urates: coagulated albumen takes the form of a white flocculent precipitate, the phosphates have the appearance of a more uniform white sediment, while the urates are readily recognisable by their yellowish-red colour.

As albumen is thrown down by heat in acid urine only, and not, or only in small quantity as compared with the actual amount present, in urine which is alkaline, the reaction of the urine should always be ascertained before boiling, and if it turn out to be neutral or alkaline it should be acidified with a drop or two of acetic acid.

Albuminous urines are seldom perfectly clear, the cloudiness by which they are pervaded being caused by the various figurate organised elements which such urines generally contain. If the turbidity be considerable, and the urine be but very slightly albuminous, it is desirable to apply the flame of the lamp to only the upper layer of fluid in the test tube, as this renders the contrast between the increased cloudiness of the warmed upper portion and the unchanged appearance of the lower portion of the urine the more apparent. If the cloud produced by heat be so indistinct as to leave it still doubtful whether or not it is really due to albumen, a portion of the recently passed urine must be filtered and the clear filtrate examined for albumen.

The daily loss of albumen in inflammatory affections of the kidneys is exceedingly variable, amounting to 5—10 grm. in the less severe cases, and 15—20 grm. in those in which the disease is of greater intensity; the urine is occasionally so loaded with albumen that almost the whole of the fluid in the tube is changed into a firm coagulum on boiling. To observe whether the excretion of albumen is on the increase or the decrease, the test-tubes containing the different specimens of urine examined from day to day should be set aside in an upright position, and the depth of the layer of precipitated albumen in each compared; this method gives, of course, only an approximate quantitative estimate. The glasses must be of the same diameter and an equal volume of urine must be used each day.

Of the various modes of calculating accurately the amount of albumen in the urine the simplest is the optical method, circumpolarization with the Soleil-Ventzke apparatus. After this mechanism has been so arranged that the zero of the nonius corresponds with the zero of the scale, a glass tube is introduced, filled with the albuminous urine, which is previously filtered and decolorized by being passed through animal charcoal. The two halves of the field, which before the introduction of the albuminous fluid appear exactly alike, at once become different in colour. The compensator is now moved, by means of the screw, towards the *left* till both sides of the field are again of precisely the same hue. The nonius shows the extent of the displacement on the scale, and from this the proportion of albumen in the fluid is known; thus, if the compensator requires to be shifted till the nonius points to 3·6 before the desired identity in colour of the two divisions of the field is obtained, the urine is said to contain 3·6 grammes of albumen in every 100 grammes. The exactness of the calculation by this method depends on the examiner's sharpness of vision.

A high degree of albuminuria is always caused by nephritis; the existence of dropsy, above all the discovery of renal elements in the urine by microscopic investigation (epithelial scales, casts of the uriniferous tubules), are also indications which point to the same diagnostic conclusion. The albuminuria usually attends the nephritic process throughout its whole course, though at times it may diminish very considerably, or in some rare cases may almost totally disappear.

Albuminuria is also observed, altogether apart from any inflammatory condition of the kidneys, in catarrhs and other severer affections of the uropoietic system, from the pelvis of the kidneys to the external orifice of the urethral canal; the presence of the albumen is then due to the admixture of pus and the corpuscles and serum of the blood. In these cases albumen is detected also in the filtered urine; generally, however, the urine is less albuminous than that discharged in pronounced renal disease.—Finally, a moderate degree of albuminuria occasionally appears, but remains only a short time, in a great many severe acute and chronic diseases; in such affections the urine, unlike that passed in nephritis, shows no figurate elements (casts, &c.,) under the microscope.

Besides albumen derived from the serum of the blood there are found, in all albuminous urines, certain other bodies of similar constitution,—paraglobulin (Edlefsen, Senator) and peptone; these are sometimes also found in the urine when it is free of ordinary albumen (Gerhardt). In *chyluria*, a disorder met with in tropical countries, there exists in the urine an albuminoid substance of a totally different nature from the albumen of the serum (Eggel).

Fibrin is invariably present in urine containing *blood;* but it is observed also when there is no hæmaturia, in the form of coagula or of *fibrinous* (granular) *casts*, in many cases of very acute and severe nephritis.

Mucus occurs in mere traces in normal urine; it is derived from the secretion of the lining membrane of the urethral canal, in women often from the vaginal secretion. Pathologically it is discovered in the urine chiefly in cases of catarrh of the bladder, and is easily recognised with the naked eye as a viscid stringy cloud which floats about in the fluid and eventually settles to the bottom of the glass when the urine has been stationary for some time. Examined microscopically it is seen to be formed essentially of a clear, absolutely structureless substance, in which, however, lie embedded large polygonal epithelial cells from the bladder and round oval *mucus-corpuscles.*

Sugar. The saccharine urine passed in diabetes mellitus has the following properties: *its quantity is very notably increased;* its *colour* is very pale or faintly yellowish with a tinge of green; it is perfectly *clear* and *limpid*, and gives no sediment on standing; its *specific gravity is unusually high*, 1030—1040, in certain cases 1050 and even higher.

There are four tests which serve for the detection of sugar (grape sugar) in the urine: *Trommer's process*, the *caustic potash*, the *bismuth*, and the *fermentation tests*; the first of these is by far the most delicate and is that most often used.

1. *Trommer's Test.* To a little of the saccharine urine placed in a test-tube are added first a few drops of a *solution of sulphate of copper* and afterwards a certain quantity of *liquor potassæ*, the latter being gently poured in till the whole fluid assumes a clear and beautiful *dark blue colour*. On obtaining this coloration the examiner may feel sure that the urine is saccharine, as no other sort of urine undergoes this change on being treated with these reagents.—On now heating the mixture an *orange yellow* or *brick red* precipitate is formed, which in a short time falls to the bottom of the test-tube. This deposit consists of *suboxide of copper*. The chemical reaction which takes place is the following: the sulphuric acid of the sulphate of copper unites with the stronger base, the caustic potash, forming sulphate of potash; the sugar is oxidised at the expense of the oxide of copper, and the latter, being *reduced* to the condition of a red insoluble suboxide, is at once precipitated.—This process also bears the name of the *reduction test*.

2. *Caustic potash test. Moore's test.* The suspected urine is mixed with a little liquor potassæ and warmed, the heat being applied to only the upper part of the fluid in the tube; the part so treated takes on a clear yellowish-brown colour, deepening, on further heating, to a *brownish-red*, which is the more intense the more potash has been added. The portion of fluid which is not heated preserves its original colour, which renders the change just described the more striking. If the warmth be kept up till the urine boils there is developed, especially on adding a little nitric acid, a sweetish smell of burnt sugar. Urine coloured brown by the caustic potash process turns still darker on keeping for a short time.

3. *Bismuth test.* To the urine should be added first a few drops of *liquor potassæ* and then as much *nitrate of bismuth* as will lie on the point of an ordinary knife, the latter reagent being an insoluble, heavy, white powder which rests on the bottom of the glass. The application of heat now imparts to the urine a clear brownish-red colour, (as in the last test), while it *blackens* the bismuth. The chemical change which takes place is simply the reduction of the bismuth salt, and is exactly analogous to that which occurs in Trommer's test: the nitric acid of the salt of bismuth unites with the caustic potash, a portion of the oxygen of the oxide of bismuth is appropriated by the sugar, and black suboxide of bismuth is left.

4. *Fermentation test.* Saccharine urine, when mixed with yeast, ferments, the sugar being changed into carbonic acid and alcohol.

The indications offered by these tests may be obscured by the presence of various substances in the urine which prevent the reduction of the copper or bismuth salt; the urine may, for instance, contain albumen as well as sugar, when the former must first be precipitated and then removed by filtering before the testing for sugar can be proceeded with.—There are several other tests for sugar in the urine, but as they are not much used no account of them need be given here.

The quantity of sugar present in diabetic urine varies from ½—10 per cent.; in the great majority of cases it fluctuates within narrow limits, from about 3—5 per cent. The per centage is calculated by using *Fehling's standard solution of sulphate of copper* or by means of the Soleil-Ventzke saccharimeter. After introducing the glass tube filled with the saccharine urine into the apparatus just named, the nonius, previously arranged so that it points to zero on the scale, is turned to the *right* till both fields have the same colour; the extent of this displacement to the right indicates the per centage of sugar in the urine.

FIGURATE ELEMENTS IN THE URINE.

Normal urine is perfectly clear, containing neither crystalline nor any other organic figurate element, except possibly now and then traces of mucus. If, however, the urine be turbid immediately after emission, we have distinct evidence of the presence of such constituents. In order to examine these with the microscope the urine must first be filtered, as they occur in very small quantity compared with the volume of the fluid in which they are diffused; or the urine should be allowed to stand some time in a glass which tapers towards the bottom, when a small portion of the sediment may be removed with a pipette.

The organic figurate elements found in the urine in disease are pus cells, blood corpuscles, casts of the uriniferous tubes, epithelial cells, and fungi.

Pus corpuscles. These are in no respect different from the colourless globules of the blood. A highly purulent urine is perfectly turbid and of a whitish-yellow, milky colour, even at the moment it is passed; if set aside for a short time the pus corpuscles are deposited at the bottom of the glass as a yellowish sediment. The pus globules retain their ordinary shape in the urine so long as this fluid remains acid or neutral in reaction; but the occurrence of alkaline fermentation converts them into a gelatinous mass which is quite devoid of structure under the microscope.

Pus corpuscles are found in the urine in greatest number in *catarrh of the bladder;* they are more or less abundant, however, in catarrh of any part of the urinary tract, from the pelvis of the kidneys down to the orifice of the urethra, being mechanically mixed with the urine in its passage outward. It is generally easy to infer from what part of the urinary apparatus the pus comes by noting the further results of objective examination and the other symptoms of the affection. If it be derived from the urethra, in men, a purulent fluid may also usually be expressed from the canal before as well as after micturition; in women pus cells often gain admission into the urine from the admixture of vaginal secretions (as in cases of leucorrhœa); to prevent error it is advisable in such circumstances to draw off the urine with a catheter. If the pus be secreted by the bladder, which in chronic cases of purulent urine is the usual source, it is accompanied by a number of the large vesical epithelial cells. If it proceed from a point considerably higher, from the pelvis of the kidney, for example, there are usually also observed the other signs of positive renal disease; the principal element in the diagnosis in such cases is the exclusion of every other part of the urinary passages as a possible source of the discharge.

Red blood corpuscles. They are found in the urine in sufficiently large quantity to impart to that fluid a distinctly blood-like coloration, only as the result of hæmorrhage (rupture of some vessel) within the uropoietic apparatus. They occasionally appear in smaller numbers, but still abundantly enough to suggest to the eye at once that the urine contains blood, without rupture of the walls of the vessels; this form of bleeding, *hæmorrhage per diapedesin,* may occur in all inflammatory conditions within the uropoietic system, particularly in very severe acute nephritis, but also in the course of chronic nephritis, when the inflammatory action is from any cause increased in violence.—The shape and colour of the red blood corpuscles as they are seen in the urine, have already been described on p. 378.

Epithelial cells. The epithelial cells found in the urine may be detached from any part of the urinary apparatus, and are met with in all affections of these parts. The renal epithelial cells, as they appear in the urine in kidney diseases, are sometimes separate from each other, at other times adherent. The urinary passages throughout their whole length are protected by several layers of pavement epithelium, disposed one over the other, forming a lining membrane which is of greatest thickness in the bladder. The vesical epithelial cells are generally large, provided with only a single nucleus (like the buccal epithelial scales), polygonal, and more or less rounded at the angles,—characters which render their recognition easy. Below the superficial pavement epithelium of the bladder are numerous smaller, nucleated, *spindle-shaped* cells, which make their appearance in the urine when the catarrh is severe enough to involve the deeper layers of the epithelial lining of the bladder.

TUBE-CASTS IN THE URINE.

Before beginning to look for these bodies, which play such an important part in the diagnosis of renal diseases, the urine should be filtered, or should be allowed to stand and deposit a sediment in a glass of suitable construction, as the casts are seldom so abundant as necessarily to be present in every single drop of the urine when agitated, as immediately after emission. One drop of the sediment from the glass, or of that which is caught on the filter, should be placed under the microscope and examined with a power of 300 diameters.

These cylinders are simply casts of the uriniferous tubules, which are washed out of the kidneys by the flow of urine. They are divided into three principal varieties: epithelial, granular, and hyaline casts. *Epithelial* casts consist of the epithelial lining of the tubuli uriniferi, expelled in a more or less complete form (desquamative nephritis). As regards the manner in which the *granular* and *hyaline* casts are formed authorities are not by any means unanimous. Whilst according to the older (and recently revived) view they should be regarded as transudation-products derived from the blood, they are described by some as due simply to degenerative change in the epithelial cells. It is generally assumed that the granular casts represent the primary form of the degeneration of the renal epithelium, and that the hyaline casts are a secondary modification resulting from the same degenerative process.

In support of the theory that tube-casts are formed by exudation from the blood, Weissgerber and Perls have recently brought forward the following arguments: the epithelial lining of tubules filled with homogeneous casts is found perfectly intact; casts are often entirely wanting in tubuli whose epithelium is already the seat of very extensive degenerative change; on raising the pressure within the renal veins in animals, by constricting the principal vein, not by closing it absolutely, as this would arrest circulation in the part, fibrinous casts are invariably found in the kidneys; on microscopic examination of such sections of the kidney no appearance is discovered at any point which suggests the idea that the epithelium is metamorphosed into casts, but there are very often seen the various stages of a process in which a simple albuminous fluid, which completely fills the lumen of the uriniferous canals, seems to be gradually transformed into casts of these tubes.

The *granular* (or fibrinous) casts are of different lengths, according as they are expelled from the uriniferous tubules uninjured or in fragments; they vary in length from ½ to 1, seldom 2 mm. or a little more, and in breadth from 0·04—0·06 mm.; they are very *darkly* granular, a feature by which they are very readily known when seen, are frequently covered by blood and pus corpuscles, and contain also a yellowish fatty detritus. In very violent acute nephritis they occur in great numbers in the urine; they are also very abundant in chronic nephritis.

Hyaline casts are strikingly pale and transparent, their outline being also made more distinctly visible by staining with carmine or aniline; they are occasionally absolutely free of any figurate element, but in other cases show here and there a little granular fatty detritus or a few epithelial cells. Between the typically granular and hyaline casts come many intermediate forms; many present the characters of both varieties, being darkly granular at one part and perfectly pale and clear at another. In length and breadth there is no difference between hyaline and granular casts.

Epithelial casts consist almost entirely of the detached epithelial lining of the uriniferous canals; the individual cells of which they are composed present generally the signs of being in a more or less advanced state of degeneration, they are dull and clouded in appearance, swollen, dotted with numerous brilliant globules of fat, and are occasionally surrounded and almost hidden by blood and pus corpuscles and fatty débris. They are, as a rule, easily distinguished from the other figurate constituents of the urine.

Tube casts in the urine are commonly accompanied by free red blood corpuscles, colourless blood globules (pus corpuscles), and epithelial cells. These elements often afford a clue to the particular stage which the morbid process within the kidneys has reached, though any inference based on the results of microscopic examination alone is apt to be fallacious. A very abundant fatty detritus mingled with epithelial cells in a state of fatty degeneration, and the presence also of large numbers of thin narrow casts, generally indicate that the nephritis has arrived at a somewhat advanced stage,—atrophy; very large, broad granular casts and red blood corpuscles are indicative of the early stages of nephritis or of a fresh exacerbation of an old-standing affection; very frequently, however, all the different kinds of casts are found together in the same urine. The special *form* of nephritis present in each case cannot be determined by microscopic examination *alone*.

Whilst in all cases the occurrence of tube-casts in the urine may of itself be accepted as conclusive evidence of the existence of an inflammation of the kidneys,—an inflammation which may be simple and uncomplicated, or which may appear in connection with other affections, such as scarlatina, diphtheria, cholera, small-pox, &c.,—the other figurate elements, the red and white blood corpuscles, epithelial cells, detritus, are to be regarded as diagnostic of nephritis only when accompanied by casts; wherever such casts are wanting the microscope alone is not sufficient to fix with precision the site of the disease.

Coagula of fibrin are often observed in the urine in acute and hæmorrhagic nephritis; these bodies are somewhat elongated in shape and are not unlike granular casts, but may be distinguished from these by the irregularity of their outline and the total absence of structure and of figurate elements.

Every urine which contains casts is albuminous. There exists no absolute relation between the quantity of the albumen and the number of the casts; the latter may be few in a highly albuminous urine, or plentiful when the albuminuria is slight. Thus, in amyloid degeneration of the kidneys the urine may be loaded with albumen but may show very few casts under the microscope, while in acute renal disorders tube-casts are often abundant in a urine which is but feebly albuminous.—Nothnagel states also that *hyaline casts* always appear in the urine in intense *icterus*.

INORGANIC URINARY SEDIMENTS.

The sediments which are met with in acid urines, both normally (but only in traces) after cooling and in pathological conditions, are the urate of soda and free uric acid, more rarely the oxalate of lime; the sediments of ammoniacal urine consist of urate of ammonia and phosphates (phosphate of lime and ammoniaco-magnesian phosphate).

Urate of Soda. This salt, which is found in small quantities in normal urine, is deposited in great abundance in all febrile diseases, especially during the critical period, after violent physical exertion, and in the urine of those who habitually indulge to excess in eating. It is precipitated only when the urine cools, and forms a dirty yellowish clay-coloured or reddish (brick-dust) sediment. Under the microscope it is seen to be *amorphous*, and is made up of very minute, irregular granules, often aggregated into small masses, but easily broken up and reduced to its original divided condition by gentle pressure with the object-glass. The opacity occasioned in the urine by the urate of soda disappears rapidly on the application of heat, the fluid becoming perfectly clear.

Uric Acid. In warm urine this substance is but very sparingly soluble; it falls at once as a crystalline sediment when the urine cools, and in being precipitated takes up a portion of the urinary colouring matter, from which it acquires a yellowish red or sometimes faintly brown coloration. Under the microscope it shows the most diverse crystalline forms, appearing usually in rhombic plates or columns, four-sided prisms, often in dumb-bell or barrel-shaped crystals. Sometimes these crystals are separate from each other, at other times arranged in groups; many of them are large enough to be visible to the naked eye, and for microscopic examination they demand at most only a low power, 100—150 diameters. In the urine it is commonly combined with the acid urates of soda and potash. On adding hydrochloric acid to the urine the uric acid is set free and in a short time crystallizes out; the process of crystallization may be observed in the field of the microscope on adding one drop of hydrochloric acid to a sediment of urate of soda.

Uric acid may be recognised by its chemical reaction as well as by its microscopic characters. Thus, on dissolving a few crystals in a little nitric acid, warming and evaporating, and treating the reddish residue with a weak solution of caustic ammonia, a deep purple coloration is obtained (purpurate of ammonia, murexid), which changes to violet blue on the further addition of a few drops of caustic potash or soda.

The excretion of uric acid is doubled, sometimes trebled, in febrile and other diseases (of the respiratory and circulatory organs, disturbances of nutrition, &c.); in arthritis it is deposited in the joints. Renal calculi are often composed exclusively or in great part of uric acid and its salts.

The oxalate of lime, a normal though by no means a constant constituent of the urine, presents itself under the microscope in the form of exceedingly minute octohedra, bearing a certain resemblance to the envelope of a letter. This deposit is generally very scanty, and is most often thrown down in urine which also contains uric acid. The abundant discharge of such crystals is not an unfailing sign of a pathologically increased oxaluria; the urine may be moderately rich in oxalic acid without giving rise to the separation of oxalate of lime crystals, as the latter salt may be held in solution, especially by the acid phosphate of soda. Its quantity per day seldom rises above 2 centigrammes (Fürbringer). It sometimes forms calculi in the kidneys and bladder.

Phosphate of lime and *phosphate of magnesia* are soluble in acids and are therefore not deposited in acid urines; they are excreted to about the extent of 1 gramme per day (Neubauer). When present in the urine in large quantity they are precipitated as an abundant white sediment, the urine being neutral or alkaline to test-paper. Phosphate of lime occurs as an amorphous powder, sometimes also in crystals, often forming beautiful star-shaped masses. The phosphate of *magnesia* is also sometimes deposited in the crystalline form as elongated plates (Stein). Increase of the earthy phosphates is noticed often in phthisis, in rachitis in children, and in other diseases, frequently keeping pace with a similar increase in the separation of indican (Senator).—*Cystine* is rather a rare constituent of the urine, only 52 cases in which it was detected being recorded in medical literature; it sometimes forms concretions in the kidneys, at other times crystallizes in colourless hexagonal plates or prisms. Among other points by which cystine may be distinguished from uric acid, which frequently assumes exactly the same crystalline forms, there is this, that the former, when dissolved in nitric acid and treated with ammonia, gives no murexid reaction. The daily excretion of cystine amounted, in the case recently put on record by Niemann, to about 0·5 gramme, and in that reported by Loebisch to 0·4 gramme.—*Leucine* and *Tyrosine* are met with in the urine in certain cases of acute atrophy of the liver and variola; the former substance appears in granular, yellowish, globular masses, or, when perfectly pure, in white and exceedingly thin plates; the latter crystallizes in very fine white needles.

Urate of ammonia is formed when the urine undergoes alkaline fermentation, crystallizing in small spherical masses studded over with spines (the thorn-apple crystals).

Ammoniaco-magnesian phosphate is invariably separated, as soon as the urine becomes alkaline, in colourless prismatic crystals of various sizes, most of them slightly modified and tending to the ordinary coffin-lid shape. These crystals are freely soluble in acetic acid, a property by which they may readily be distinguished from the oxalate of lime, which is not soluble in acetic acid; this is a reaction of some importance, as the phosphatic crystals, when very small, resemble closely those of the oxalate of lime.

Of the lower organisms found in the urine should be noticed the *rod bacteria* which always accompany fermentation of the urine, and *sarcinæ*.

VOMITING.

When the ramifications of the pneumogastric nerve in the mucous membrane of the stomach and pharynx are subjected directly or indirectly to any abnormal irritation, reflex and very energetic contraction of the diaphragm and abdominal muscles is set up, whereby the stomach is firmly compressed on all sides and its contents discharged upwards. The stomach itself is not *actively* concerned in the act of vomiting, except in so far as it contributes by the opening of its cardiac orifice; its muscular coat contracts very little, if at all.*

The irritation of the sensory nerve-terminations in the stomach may be direct or indirect. *Direct* irritation is produced by emetics, poisons, nauseous substances, violent shocks, or by merely overloading the stomach; in all gastric diseases, from simple catarrh to the malignant new formations, and often also in simple hyperæsthesia of the gastric nerves, unaccompanied by any anatomical change, the stomach is more or less directly irritated. There is no disease of the stomach in which vomiting, transient or persistent and recurrent, may not appear as a symptom in some part of its course, though in many cases it is entirely wanting from beginning to end; it is only in cancer, especially when it causes stricture of the pylorus, that vomiting comes to be an absolutely constant symptom.

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* It is necessary that the cardiac orifice of the stomach should be opened before vomiting can take place. The abdominal pressure alone is not sufficient to effect this, as there are certain conditions, difficult and painful defæcation, for example, in which, notwithstanding the amount of pressure brought to bear on the stomach by the abdominal muscles, the contents of the stomach are not ejected, simply because the cardiac orifice of the organ remains closed; on the other hand, vomiting is not unfrequently observed in animals on exposing the stomach and injecting tartarated antimony into the veins, though in this case the force exerted by the walls of the abdomen is completely eliminated as a factor in the process.

In diseases of the stomach vomiting takes place more readily when the organ is full than when it is empty; it is occasionally brought on also in such circumstances by every variety of solid food, sometimes even by the blandest fluids, at other times only by taking things which are difficult to digest or by special articles of diet; it may occur immediately after eating or only after the lapse of some time. In certain cases it is possible to form some idea as to the seat of the disease from the interval which elapses between the taking of food and the starting of the vomiting; thus, in cancer of the stomach the patient rejects his food almost directly after a meal when the cardiac orifice is contracted, but if the disease be located about the pyloric end, producing stenosis of the pylorus, the vomiting does not begin till several hours have passed.

The irritation of the gastric nerves may be *indirect*, the organ itself being perfectly healthy in structure. This is sometimes due to abnormal excitation of the vagus at its origin in the brain,—hence the frequency of vomiting in cerebral diseases and in certain affections of the nervous system,—or to reflex irritation of the vagus through some of the abdominal plexuses of the sympathetic, with which it is connected by anastomoses. In some cases the vomiting which so often attends affections of the abdominal organs may be explained by the relation which subsists between these two portions of the nervous system; in many other cases, however, this explanation is not so satisfactory.

EXAMINATION OF THE VOMITED MATTERS.

This is generally made with the naked eye, which in most instances is quite sufficient for diagnostic purposes; in certain circumstances, however, a microscopic examination becomes necessary.

According to the stage of digestion at which vomiting has occurred, the matters brought up, consisting partly of fluids and partly of solids, are more or less acted upon by the gastric juice, and have a more or less powerful acid odour. When the stomach is empty, or after repeated evacuation of its contents, vomiting expels only a ropy, viscid, mucous secretion, with which are sometimes mingled biliary matters from the duodenum, which give to the discharges a greenish coloration. The bile is pressed out of the duodenum into the stomach by the energetic contraction of the abdominal muscles, particularly when the vomiting is severe and accompanied by much straining,—as it always is when the stomach is empty.

Of the abnormal elements which may present themselves in the vomited matters the most important is *blood*. In some cases the proportion which appears among the vomited materials is small, in others the blood is rejected nearly pure, almost always coagulated and blackened by the action of the acids of the stomach, when it resembles black coffee-grounds. Hæmatemesis is observed most often in the ulcerative affections of the stomach, and is perhaps most abundant in round ulcer; the bleeding is very free also in the ulceration which is due to corrosive poisons. Hæmorrhage may take place even in the absence of any anatomical lesion of the stomach, from congestion of the gastric veins, from engorgement of the portal system (as in cirrhosis of the liver), or from overloading of the gastric vessels from other causes; of this last-mentioned variety is the periodical recurrent hæmatemesis met with in certain cases of amenorrhœa.

Sarcinæ are frequently found in the vomited matters in chronic gastric catarrh, and particularly in cases of dilatation of the stomach. They are developed in all conditions in which the food remains for an unduly long period in the stomach and in this way undergoes various abnormal modifications (fermentation, &c.).

Sarcinæ consist of small four-sided cells, measuring about 0·01 mm. in diameter, which are usually united in fours to form a single element; these elements, again, often combine similarly in fours to form still larger squares presenting sixteen separate subdivisions; all further increase also is made in fours.

Ascarides lumbricoides sometimes find their way from the bowel into the stomach, and may then be mingled with the vomited matters. Besides these abnormal constituents, microscopic examination reveals many other substances derived from the food taken, all more or less altered in appearance by the action of the gastric juice.

THE INTESTINAL DISCHARGES.

Alteration in the frequency and in the appearance of the alvine discharges are the principal objective signs of functional intestinal disturbance. The motions may be fewer than normally (*constipation*), or more frequent and liquid (*diarrhœa*).

Constipation.

Slight constipation, in which the bowels are moved about every two days, and a severer form also in which the interval between the motions is prolonged to 3—4 days, are very common phenomena; in many instances such a disorder is periodical in its recurrence, and in some persons habitual. Constipation lasting 5—6 days is seldom met with, as therapeutic measures intended to remedy the condition are generally adopted before it has continued so long, while still longer intervals, 2—3 weeks, for instance, are very exceptional.

Constipation is most commonly to be ascribed to *sluggish peristaltic action of the bowel*, and in a smaller number of cases to the presence of some *obstruction within the bowel*, which mechanically *opposes the onward progress of the intestinal contents*, and which may, in certain circumstances, offer an absolute barrier to their passage.

The *peristaltic movements* of the bowel are rendered *sluggish* by very many different causes, which are in part connected with the manner of life of the individual, and which in the more marked forms of the affection are invariably of a pathological nature. These causes may be grouped together in the following way: want of active physical exercise, slowness of digestion and tissue-metamorphosis, unsuitable diet (consisting especially of very solid and unstimulating substances), distension of the bowel by gas; peristaltic action is slackened also when bile, a normal stimulant of the bowel, is wanting in the intestinal contents, a fact which explains the frequency of constipation in obstruction of the bile-duct, from duodenal catarrh and other causes; the muscular coat of the bowel may, further, be temporarily enfeebled through over-exertion and simple fatigue, induced by severe diarrhœa or the action of medicines,* or it may be permanently weakened by repeated drastic purgatives; † the peristaltic movements are also retarded by diminution in the contractile power of the muscular tunic of the intestine, such as that observed in inflammation of its peritoneal investment, or by deficient innervation, as in diseases of the brain and spinal cord, perhaps also by spasmodic contraction of the intestinal muscles and consequent narrowing of the alimentary canal, as in lead colic.—In some cases several of the above-named causes may be in operation simultaneously; their recognition is usually a matter of no difficulty.

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* When in disease the peristaltic movements of the bowel become abnormally active they may be moderated by the use of remedies which soothe the irritability of the sensory nerves of the intestinal mucous membrane or which diminish the secretion from the internal surface of the bowel; to a slight degree also this may be accomplished by a properly-regulated diet, consisting of bland mucilaginous substances which cover and protect the sensitive mucous surface.

† Many who suffer from habitual constipation aggravate their malady by the misuse of drastic purgatives, the effect of which is that an ever-increasing degree of irritation, obtained either by adding to the dose or by the employment of more energetic drugs, is needed to awaken peristaltic action in the bowel; the natural stimulus, the mere presence of the intestinal contents, is of very little avail in such cases, though its influence is soon in great part restored on withholding the purgative.

The onward movement of the contents of the bowel may be rendered slower or may be absolutely arrested, and constipation of various degrees of severity, or even impassable obstruction, may be set up, by the presence of some *mechanical obstruction* which compresses or completely closes the bowel; constipation, however, is much more seldom due to this condition than to feeble peristaltic action. This encroachment on the lumen of the intestine is dependent on causes some of which lie within, others without the bowel. Among the *internal* causes should be reckoned strictures, invaginations (intussusceptions), twisting and flexion of the intestine; large, hard fæcal masses, and large biliary calculi, lodged at various points in the intestinal canal, particularly above strictures, where they distend the bowel and form diverticula, constitute mechanical obstacles of the character described. To the group of causes *external* to the bowel belong compression by the uterus in the gravid state or when pathologically enlarged or retroverted, by ovarian tumours, by very marked hypertrophy of the prostate, and by strangulation of the intestine (strangulated hernia). If these obstacles produce merely a narrowing of the lumen of the intestine, they may always be overcome by drastic purgatives, by exciting powerful peristaltic action; but if the closure of the canal be complete, as in strangulation, no advance of the retained intestinal contents is possible,—they rather travel backwards in the bowel if no operative measures be adopted for the removal of the obstruction, and eventually, on reaching the stomach, are discharged by vomiting. This (stercoraceous) form of vomiting is often observed in ileus.

DIARRHŒA.

Diarrhœal discharges are more fluid and are usually also more abundant than those of health. It sometimes happens that a person is seized with a sudden but transient attack of diarrhœa, one or two thin watery motions following closely one on the other, the disturbance then subsiding and stools of natural consistence making their appearance; this is usually caused by some short-lived irritation of the intestinal mucous membrane by errors in diet, by cold in those who are so predisposed, by sudden change in the habits of life, &c. Wherever actual disease of the mucous membrane has given rise to diarrhœa the frequency of the motions is invariably increased; 3—4 evacuations of the bowels in the 24 hours are a moderate number in acute intestinal diseases, while in the intestinal catarrh of children, in typhoid fever, dysentery, and cholera they may number 20 or more in the 24 hours. This stage of the affection, however, in which the stools are so loose and so frequent, does not last long,—a few days or even a shorter time. In chronic diseases of the intestine diarrhœa is never, except incidentally, so severe as in acute diseases; occasionally, indeed, it is varied by longer or shorter periods of constipation.

Diarrhœal evacuations are sometimes perfectly painless; at other times they are preceded by a peculiar painful sensation, known as *colic*, which generally originates about the middle of the abdomen and radiates towards the other parts. Painful and painless stools may alternate with each other in the same patient; the presence or absence of pain therefore, in the majority of cases at least, offers no diagnostic indication of the special form of disease in which the bowel is involved. Nevertheless pain before or at stool, if associated with tenderness to pressure on the abdomen, generally warrants the inference that the bowel is probably the seat of an ulcerative affection.

The alvine discharges always assume a diarrhœal character when from any cause the *peristaltic movements* of the bowel are *increased* in rapidity and energy, so that the intestinal contents are carried more quickly onward, so quickly that they are expelled before their fluid constituents (dissolved alimentary matters and fluid derived from the intestinal vessels) have been to a sufficient extent absorbed.

The *causes of the increased peristaltic action* are many and varied, but they all admit of one of two interpretations,—the sensory nerves of the intestinal mucous membrane are either subjected to abnormal irritation or they are unduly sensitive; both classes of causes are frequently present. The mucous membrane is irritated by various articles of diet, particularly if they are taken in large quantity or injudiciously combined. The lining membrane of the bowel becomes abnormally sensitive when it is attacked by disease; all intestinal affections, from simple, transient catarrh to the severe ulcerative processes, are usually accompanied by diarrhœa, which in chronic diseases may for a time disappear and be replaced by marked constipation. This increased sensibility may in acute intestinal diseases become so intense that even the intestinal secretions, which in such cases are greatly augmented in quantity, act as powerful irritants.

Peristaltic movements, excited in any part of the intestine by any of the above-named pathological causes, travel onward in undiminished intensity through the whole length of the bowel, so that, for instance, in catarrh of the small intestine they may be transmitted from their starting-point there to the perfectly healthy colon.

If the increased activity of the peristaltic movements in catarrhs of the small intestine were limited to this portion of the alimentary tract, the movements of the large intestine continuing normal, the intestinal contents would remain long enough in the colon to permit of the absorption of their fluid constituents, and the properly diarrhœal discharges would be wanting,—which, however, is not the case.

The loose diarrhœal motions also which follow the therapeutical use of drastic purgatives, are produced by the increased energy of the peristaltic movements.

EXAMINATION OF THE INTESTINAL DISCHARGES.

In examining the motions attention should be directed specially to the following points: their quantity, consistence and form, colour, odour, and the presence in them of abnormal constituents.

The *quantity* of fæcal matters passed in health depends on the amount of food taken and on the proportion of indigestible elements which the various articles of diet contain. It is always increased in diarrhœa, by the addition of the intestinal secretions and of such portions of food as have escaped absorption. In acute intestinal diseases,—acute catarrh, European and Asiatic cholera, dysentery, for example,—the evacuations may be enormous, consisting in great part of watery transudation from the mucous surface and to but a slight extent of excrementitious matters.

The *consistence* of solid motions is proportionate to the time taken by the intestinal contents in travelling through the bowel to the rectum; the slower the rate of transmission the more completely will their fluid constituents be removed by absorption and the firmer will be the fæces. In those who suffer from constipation, accordingly, the motions are usually hard and dry. The *form* of the motions depends on their consistence; only such as are solid are "formed", semi-solid discharges being described as pultaceous, those which are more fluid as soup-like, &c.

The *colour* of the dejecta, which in health is brown, is owing to the admixture of the colouring matter of the bile, hydrobilirubin. When the bile is prevented from entering the bowel, as when the ductus choledochus is closed, the motions have a dirty greyish, clay colour.

It is only in duodenal catarrh that discharges absolutely destitute of bile are seen, as it is only in this affection that more or less complete closure of the bile duct takes place; in diseases of the liver and biliary ducts, though the flow of bile into the duodenum is often impeded it is never entirely arrested. If, in the course of a duodenal catarrh, the fæces begin to show again a brownish coloration, it may with confidence be inferred that the bile is again escaping into the duodenum even though the jaundice show no sign of abating.

Diarrhœal motions also, like those of semi-liquid consistence, are yellowish-brown, but turn clearer and paler the more abundant they become, as the bile which enters the duodenum is diffused through a much larger mass of fluid. If the discharges be exceedingly profuse, as in Asiatic cholera, the quantity of bile poured out remaining the same or even being reduced, their yellow colour is lost by degrees till eventually they exhibit no further trace of the presence of bile; they are then perfectly colourless or dirty white, and are composed only of the rice-water fluid exuded from the intestinal vessels.

In young infants the motions, which normally are of a clear yellowish brown colour, often become greenish, especially when dyspepsia is present; the cause of this coloration has not yet been satisfactorily made out, though it is probably to be sought in a metamorphosis of bilirubin into biliverdin. The dejecta acquire a similar grass-green hue on the internal administration of calomel.

The motions take on an abnormally deep brown colour in constipation of long duration. They are blackened by the internal use of the preparations of iron, of which only a small portion is absorbed, the remainder being mixed up in the intestinal contents.

Fluid motions are stained reddish-brown or brownish-black (chocolate-colour) when they contain large quantities of blood derived from the upper part of the intestinal canal; in such cases the oxyhæmoglobin of the arterial blood is transformed into methæmoglobin and hæmatin. When the blood comes from the rectum it does not remain long enough in contact with the fæcal matters to permit of this round of changes beginning, so that the colour it imparts to the motions is a clear, well-marked red. The presence of even a slight trace of blood is indicated by the colour it communicates to the evacuations.

The *odour* of the motions is characteristic; it is due essentially to the volatile products formed in connection with the decomposition of fatty matters. It is of very little practical importance, as the differences which it presents are only differences in intensity, not in kind. The simpler and plainer the food, as in infants still nursed at the breast, the less powerful the smell. When the motions are very fluid and succeed each other rapidly their odour diminishes very considerably. The rice-water discharge observed in cholera is almost entirely devoid of odour, as it contains practically no fæculent materials. In inflammatory and ulcerative affections of the bowel, particularly of the rectum (cancer, for example), the motions sometimes have an extremely offensive and penetrating smell.

ABNORMAL SUBSTANCES IN THE INTESTINAL DISCHARGES.

The appearance of any abnormal constituent in the motions is, as a general rule, of practical or diagnostic importance only when the admixture of the foreign element is so large as to be plainly obvious to the naked eye. Occasionally, however, the presence of the morbid material cannot be satisfactorily determined without having recourse to microscopic examination.

The abnormal substances found in the alvine discharges, recognisable usually by simple inspection, are the following:

Blood. Motions which contain a large proportion of blood are more or less fluid, as the diseases which give rise to intestinal hæmorrhages are also usually accompanied by diarrhœa. Smaller quantities of blood may be mingled with fæculent matters of a semi-solid consistence or disposed in streaks on the surface of the motions. Blood may also be voided perfectly pure and unaltered. In certain circumstances the blood which is seen in the stools comes from the stomach, as, for instance, when the blood poured out in cases of perforating gastric ulcer is not got rid of by vomiting but passes downwards through the intestine; such discharges commonly take the form of masses of coagula of a brown or blackish-red colour, resembling tar. In all other cases the blood is derived from the intestine, such hæmorrhage being caused usually by ulceration of the mucous membrane; blood is therefore very frequently present in the evacuations in typhoid fever and dysentery.—But the existence of an actual lesion of the mucous membrane of the bowel is not necessary to the appearance of blood in the motions; the hæmorrhage may take place from ruptured capillaries or from the bursting of the dilated and tensely engorged veins of the rectum. It is to the occurrence of bleeding of the latter variety that the disease known as hæmorrhoids owes its name.

But the dilated hæmorrhoidal veins are not the only source of intestinal hæmorrhage; in those cases in which circulation through the liver is impeded all the abdominal veins which join the portal system, and among them those of the rectum, are overcharged with blood, and in this way intestinal vessels of small calibre are often ruptured. Hæmorrhage from this cause is not unfrequently observed in cirrhosis of the liver and in cases in which the portal vein is compressed by tumours.

Pus is found in the motions, or is passed in small quantities pure and unmixed, in ulcerative affections, particularly of the rectum, and in chronic rectal inflammations; or it may come from abscesses situated close to the rectum and opening into it.—*Mucous secretions* also, usually mingled with pus, are discharged in various rectal diseases.

Tissue-elements from the intestinal mucous membrane occur in the stools in violent enteritis and ulcerative affections of the bowel; they are composed for the most part of detached epithelium, sometimes of shreds of mucous membrane. The intestinal epithelium is separated freely in cholera.

Fungi appear abundantly in the rice-water motions in cholera. In the intestinal or infectious disease designated mycosis intestinalis, to which attention has recently been directed, innumerable fungous elements and bacteria have been discovered not only in the dejecta but also in the wall of the intestine, in various other tissues, and in the blood.[*]

The motions, particularly those observed in the diarrhœa of children, often contain fragments of undigested food.

Worms are frequently voided with the intestinal discharges, sometimes spontaneously, but usually, and in greatest number, after the administration of anthelmintics. The eggs of the worms are often expelled with the fæces, occasionally in such abundance that several may be seen in every drop examined under the microscope.

The worms which inhabit the human intestine are: the *ascaris lumbricoides*, the round worm, whose habitat is the whole of the small intestine; the *oxyuris vermicularis*, the thread-worm, which lodges in the large intestine, chiefly in the rectum; both of these are most commonly found in children. The *tricocephalus dispar*, the whip-worm, lives in the cæcum and colon, and occurs in both children and adults. There are three varieties of tape worm, *tænia solium*, *tænia saginata* (or *mediocanellata*), and *bothriocephalus latus;* these parasites are met with principally in adults, and occupy the small intestine. Bothriocephalus latus is rare in Germany, and is seldom observed in countries in which tænia solium abounds.

Calculous formations, (*gall-stones and intestinal calculi*), occasionally appear in the motions. The former may be detected in the fæces after the patient has suffered for a longer or shorter period from gall-stone colic; but small calculi, biliary gravel, very often escape observation. Gall-stones enter the duodenum through the ductus choledochus, or sometimes through openings formed between the gall-bladder and the bowel by inflammatory adhesion and subsequent perforation, a process which explains satisfactorily the voiding of concretions so large that they could not possibly have passed through the common bile-duct. Biliary calculi are composed chiefly of cholesterine, and are therefore soluble in alcohol and ether.—Intestinal concretions are formed in the bowel itself, certain salts, particularly phosphate of magnesia, ammoniaco-magnesian phosphate, and various organic combinations, being accumulated round some solid body (such as a fruit-stone) as a centre; in some cases organic concretions, blood clots, undigested fragments of food, or very hard fæcal masses, serve as the nucleus round which the different constituents of the stone are deposited.

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[*] According to all the latest observers (Buhl, Waldeyer, E. Wagner, Leube and Müller, &c.) this disease is simply a local manifestation of that known as splenic fever.

APPENDIX.

LARYNGOSCOPY.

THE larynx is explored by bringing its different parts into view by means of mirrors. Its interior is illuminated with natural or artificial light reflected downwards from the pharynx, the image of the vocal cords and the adjacent structures being received on a plane mirror (the laryngeal mirror) placed against the uvula.

The real originators of the practice of laryngoscopy are Türck and Czermak (1857—1858); the former invented the laryngeal mirror, while to the latter we owe the publication of the first experiments made with it and the introduction of artificial light as a means of illumination.[*]

The larynx may be illuminated in one of three ways: by the rays of the sun, by the diffuse light of day, or by lamp-light.

1. The full *light of the sun* is the most intense; if therefore one has at disposal a chamber into which it shines strongly its rays, either direct or reflected, may be employed. The direct solar rays are available only when they do not enter the room too obliquely. If the examination is protracted the apparent alteration which takes place in the position of the sun renders it necessary to be continually shifting the patient about. These difficulties, which at most are but trifling, may easily be got over by placing a plane mirror fitted with a universal joint (like that of the ordinary toilette mirror) in the window of the apartment, in such a position that the beams of the sun fall upon it and are thrown either directly into the patient's mouth or on a

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[*] The discovery that the interior of the larynx may be seen reflected in a mirror laid on the uvula belongs really to the early part of the present century, but the observations then made were very imperfect :—even the views of Garcia, the singing-master, on the movements of the vocal cords in phonation, published in 1855, did not materially advance the practice of laryngoscopy.

concave mirror and thence into the pharynx. The mirror must naturally be turned from time to time, to follow the apparent movements of the sun.

2. *The diffuse light of day* is strong enough to be used for the illumination of the larynx in a well-lighted room and on a clear day. It has this advantage, that it shows the interior of the larynx in its natural colouring. The best method of employing it is to reflect it into the mouth of the patient from a concave mirror of 26—29 ctm. focal distance placed so as to receive the light entering by the window. The glare may be lessened by allowing the light to pass through only a portion of the window or through a small chink in the closed shutters, but this is seldom necessary, nor is it practicable without great inconvenience. The illumination so obtained is not, of course, so bright as that produced by the rays of the sun, but it is generally sufficient to permit the several parts of the larynx to be distinctly seen. The position of the reflecting mirror for diffuse daylight and for sunlight is the same as that specified below as requisite for artificial illumination and the same apparatus is necessary.

3. *Artificial Illumination.* Sunlight and daylight being naturally available only at certain times, some method of illuminating the larynx artificially must often be resorted to, and since the introduction of laryngoscopy the efforts of inventors have been persistently directed to the perfecting of the apparatus by which this may be most efficiently accomplished.

Artificial light is used in two ways.

a. A concave mirror of about 16 ctm. focal distance, fixed on a stand, is placed on a table in front of a common lamp (either a gas, petroleum, or oil lamp), the light from which is thus reflected into the patient's mouth. By this means a very bright light is obtained; the area illuminated is indeed small, but is sufficiently large for the purpose, as only the laryngeal mirror, which projects the beam downwards into the larynx, needs to be lighted up, and not the whole of the pharynx. If it is desired to explore the mouth and pharynx, it is necessary merely to turn the concave mirror slightly from side to side in order to throw the light on the different parts one after the other.

The form of the lamp is perfectly immaterial. The concave mirror should be mounted on a stand (Türck) which is fastened to the table (Waldenburg, Bose, &c.); this is a more convenient arrangement than that in which the mirror is tied on the head of the observer by a band passing round the forehead, or is fixed in a spectacle-frame.—The concave reflector is provided with a central opening, through which the image is seen in the laryngeal mirror.

b. The *second* method of employing artificial light consists in the introduction of *lenses* between the flame of the lamp and the concave mirror which reflects it; some use only one simple biconvex lens (Levin, B. Fränkel), others two lenses (v. Bruns), or even three (Tobold).

The light obtained by this apparatus is no brighter than that given by simple reflectors without lenses, but it appears clearer, as it is distributed over a larger surface and falls on the parts in the form of a clearly-defined circle.

The manner in which the lenses are arranged and the construction of the different apparatuses, cannot be described here in detail. Tobold's apparatus is made in two forms, a larger and a smaller, both of which are fitted in a case and are easily carried about; the smaller model is in very extensive use.

The lamp should always stand to the left of the examiner, a little to the patient's right. The lamp and reflector,—and the lenses, if they are used,—must be so placed that the flame is on a level with the patient's mouth; particular care must be exercised in adjusting the various parts of the apparatus if lenses are interposed between the lamp and the mirror.

The only other instrument which is required in examining the larynx is the *laryngeal mirror*. This is a plane mirror, round, oval, or square in shape, the circular form being the most generally useful; its diameter varies from 1⅓ to 2⅓ ctm. The larger size, which has the advantage of showing a larger portion of the larynx, may be introduced when the fauces are wide, and in individuals in whom its frequent application has rendered the parts tolerant of its presence.—The mirror is made of highly polished glass, and is fastened to a stem of German silver at an angle of about 45 degrees; this stem fits into a wooden handle, in which it is firmly fixed by means of a screw.

LARYNGOSCOPIC EXAMINATION.

The patient, with his head slightly thrown back, should be seated facing the observer and as close to him as convenient. The fauces are then to be illuminated, in the manner described above, and carefully examined to determine in the first place whether they are the seat of such affections as pharyngeal catarrh, enlargement of the tonsils, ulcerations, &c., which often give rise to symptoms referred by the patient to the larynx. The examination of the larynx is then proceeded with.

To introduce the laryngeal mirror the mouth must be opened widely and the tongue thrust forward. By the latter action the cavity of the mouth is enlarged, especially posteriorly, and as it also raises the larynx somewhat the latter is more readily illuminated. The patient should put out his tongue and grasp its tip with the forefinger and thumb of his right hand, protected by a napkin, and keep it fixed in that position; if frequently examined he soon learns to keep out the tongue without the aid of the hand. In the case of patients who are still unaccustomed to the process the observer may, with the thumb and index finger of the left hand, have to catch hold of the tongue and at the same time steady the chin, in order to keep the head erect.

Many patients bear laryngoscopic examination exceedingly well; others with extreme difficulty, and only after long practice. The principal obstacles to be overcome are the disposition to elevate the tongue (and so obstruct the view) and the sensibility of the uvula to the touch of the laryngeal mirror. In most people there exists the inclination, when the tongue is thrust forward, to raise its base and press it against the hard palate, an action which renders laryngoscopy difficult or even impossible. If the patient is unable to control this movement he should be directed not to put out the tongue; if, notwithstanding this, the root of the tongue still rises, the examination may be facilitated by causing the patient to take several deep inspirations, the effect of which is usually to depress the tongue; or the base of the tongue may be kept down by means of a spatula. Occasionally these proceedings cannot be borne by the patient, on account of the choking sensations and retching they are apt to excite; repeated manipulation, however, generally lessens the sensibility of the parts.

The difficulties arising from a faulty position of the tongue having been overcome, others appear on attempting to use the laryngeal mirror, as simply touching the uvula is often enough to cause spasmodic contraction of the muscles of deglutition and severe retching. These accidents are often due to the unskilful introduction of the mirror, and patients who have frequently submitted to examination soon come to know, by the occurrence of retching, when the mirror is not properly laid against the base of the uvula, as they can bear its presence even for several minutes without discomfort if it be held so as to lie in contact only with the uvula and with none of the neighbouring parts. Some patients never get accustomed to the irritation of the mirror even though treated carefully and perseveringly, or at most they learn to endure it for a short time only. It is frequently observed also that whilst in the first few trials the mirror is borne easily and for a moderately long time, it on subsequent occasions produces an increasing amount of annoyance and at last ceases to be tolerated, particularly if the examinations be too protracted or repeated too often; then, of course, all attempts to get a view of the larynx must be given up for a time.

Besides these choking sensations and the retching just mentioned certain other difficulties, due to local morbid conditions which seriously interfere with the proper adjustment of the laryngeal mirror, are encountered. *Hypertrophied tonsils* and an *abnormally long uvula* present obstacles of this kind, the point of the latter being always apt to come in front of the mirror; in the first case the introduction of a long narrow mirror, in the second the employment of one of larger size than usual, will enable the observer to see the larynx. Mucus, when accumulated in quantity in the pharynx, may also prove troublesome; it may be removed by gargling or coughing.

Before introducing the laryngeal mirror it should be warmed, face downwards, over the globe of the lamp or, when examining by sunlight or the diffuse light of day, over the flame of a spirit lamp; to make sure that it is not too hot, and in order to avoid burning the patient, its metallic surface should then be applied to the skin of the back of the hand. The warming prevents the condensation of the moisture of the breath on the face of the mirror, and the consequent obscuration of the image it reflects, as well as the irritation which a cold body in the mouth would certainly produce. The mirror must be cleaned and warmed anew after each application.

The examiner should take the mirror in his right hand, as one holds a pen, grasping it at the point where the stem joins the wooden handle; he then passes it into the mouth, with the reflecting surface directed downwards and forwards. If he has to use his right hand, either in operating in the larynx or in manipulating a brush dipped in some astringent solution, the mirror may be introduced with the left hand. The mirror is now carried steadily backward, without touching the back of the tongue or the hard palate, and is gently laid with its metallic surface upwards against the base of the uvula, the patient being at the same time directed to say "eh" or "a" (as in *tell* or *tale*); the image of the interior of the larynx will then be seen in the mirror. The whole of the interior of the larynx does not become visible at once; to obtain a view of all its parts in succession the mirror must be turned a little to the right or left, upwards or downwards, as required. The precise direction in which it must be turned when it is desired to examine any particular part of the larynx, will be indicated on a subsequent page; but it is not always possible to follow set rules,—the method of procedure must to a great extent be determined by the special local conditions in each case, the skill of the observer being shown in the readiness with which he adapts himself to these. During all these manipulations, however, the mirror must not be moved from its position against the uvula.

The parts seen reflected in the mirror, in the order in which they appear, are: the *back of the tongue*, with its circumvallate papillæ, the anterior surface of the *epiglottis* with its three glosso-epiglottidean ligaments (l. medium and lig. lateralia), then the *arytenoid cartilages*, the *posterior part of the rima glottidis*, the *vocal cords*, and the *posterior wall of the larynx*. If the mirror be slightly lowered and pointed more nearly vertically downwards, the *anterior* wall of the larynx comes into view, the *anterior* part of the vocal cords and their angle of union anteriorly, the *false vocal cords*, the *ventricles of the larynx*, a small portion of the *posterior* surface of the epiglottis, and, if the illumination be good and the glottis widely dilated, the *trachea*, sometimes as far down even as its bifurcation. As soon as the mirror has been placed in position the first thing that should be done, particularly if the examination has to be made somewhat hurriedly, is to look for the true vocal cords; the other parts may then be inspected in succession by slightly turning the mirror as before described.

The parts of the larynx seen to the right and left in the mirror correspond with the patient's, and not with the examiner's, right and left; thus, those which appear to the observer's left in the mirror represent parts situated on the patient's right, and *vice versâ.* It is more difficult at first, however, to realise that according as the mirror is held the position of the different parts of the larynx will seem to vary. As it is usually placed, pointing obliquely downwards and backwards, the structures at the back of the larynx are seen at the bottom of the mirror (the arytenoid cartilages, for instance), and those at the front of the larynx (the anterior angle of the glottis, for example), in the upper portion of the image. A little practice soon enables one to recognise the various structures in the mirror and to refer them to their real position in the body.

EXAMINATION OF THE DIFFERENT PARTS OF THE LARYNX.

When the mirror is laid against the base of the uvula the first object seen is the *epiglottis;* on raising the mirror a little and placing it at the junction of the hard and the soft palate, the *back of the tongue* also becomes visible. The ligaments which pass from the base of the tongue to the epiglottis, the median ligament and the two lateral frænula, next catch the eye, and between these folds the two glosso-epiglottidean *sinuses* or *valleculæ.*

The *epiglottis* is the first structure which comes into view on getting the laryngeal mirror into position, only its anterior surface, however, being then seen. On the configuration of the epiglottis depends to a great extent the ease or difficulty experienced in examining the larynx. There may thus, apart from the great differences it shows with regard to size, be considerable irregularity of its anterior surface and in the outline of its anterior margin. Very frequently it is found to be inclined too far backwards, while occasionally it is markedly contracted in the middle. In both cases a large part of the larynx is shut off from inspection, and it becomes difficult, sometimes impossible, to obtain even a glimpse of the parts in the front of the laryngeal cavity (the anterior portion of the vocal cords and their angle of junction). The mirror must in such cases be thrust as deeply as possible into the pharynx, being held no longer obliquely but pointing almost vertically downwards.

When the mirror is placed in its ordinary position only the *anterior* surface of the epiglottis is exposed; to bring into view its *posterior* surface the mirror must be directed very obliquely downwards and backwards, though even then only a *portion* of this side can be seen. While the mirror is so held also the anterior part of the glottis is generally at the same time visible.

After having sufficiently investigated the parts at the base of the tongue and the epiglottis, attention should be directed to those within the larynx. On depressing the mirror and turning it somewhat more obliquely backwards the *arytenoid cartilages* next come into view. These bodies appear as two cartilaginous prominences, about as large as small peas, pale reddish in colour, and converging towards each other. In inspiration they fall widely apart, in expiration they approach each other, while in pronouncing the vowel-sound "eh" or "a" (as in *tell* or *tale*), they come into close apposition with each other. On their apices are situated the *cartilages of Santorini*, two cartilaginous nodules which are visible only when the illumination is exceptionally good.

During respiration there remains between the arytenoid cartilages a fissure of greater or less size, occupying the long diameter of the larynx, the *rima glottidis*, through which the posterior laryngeal wall is discovered. This triangular aperture is dilated in inspiration and contracts in expiration. The wider it is the more fully not only the posterior but also the anterior wall of the larynx is opened to the view; the patient should therefore always be asked to make several deep inspirations, but to do so as quietly as possible.

The rima glottidis is bounded on each side by the vocal cords. The posterior part of the vocal cords is usually seen when the arytenoid cartilages appear in the mirror, or in uttering the vowel-sounds described above. To recognise readily the vocal cords and to examine them carefully in their whole length, from their posterior attachment to the arytenoid cartilages to the point at which they join anteriorly, are the principal objects of laryngoscopy. For prolonged and thorough examination of the vocal cords, such as that often made simply for practice, quiet respiration is most favourable, occasionally interrupted only by the production of the vowel-sound "eh" or "a."

The *vocal cords* attract the attention at once, their *tendinous lustre* and *whiteness* being quite distinctive; in quiet respiration only their inner margins, which bound the rima glottidis, are observed, but in phonation, in producing the sounds mentioned above, they become visible in their whole breadth, as they are thus made to approach each other and close the glottis.

The *vibration* of the vocal cords, both of their whole breadth and of their edges alone, may be studied while the patient sings, the cords vibrating in their entire depth when the lower notes are sounded, and only their edges when the higher notes, particularly those which constitute the falsetto voice, are sung. In those who have become accustomed to examination the increase and decrease of these vibrations may be followed closely while the patient sings up and down a scale.

The appearance of the vocal cords is practically the same throughout their whole extent; at one point only do they present a yellowish spot, the *macula flava*, about as large as a pin's head and situated on their inner edge, close to the arytenoid cartilages; these spots are small nodules of fibro-cartilaginous tissue, seated on the posterior surface of the cords and shining through them.—Whilst the posterior part of the vocal cords is always easily enough seen it is generally difficult to get a good view of their anterior part, and particularly of their anterior angle of junction. If the epiglottis be markedly trough-shaped, or curved so as to resemble the Greek letter omega, a very small portion only of the larynx is open to inspection, and it is seldom possible to get a sight of the point of insertion of the cords. Even these disadvantages may sometimes be overcome, however, by securing good illumination and by varying the position of the head and of the mirror to meet the special local conditions which complicate each case. No precise set of rules can be formulated on this subject, but it will in general be found to be of service to tilt back the patient's head considerably and to push the mirror more deeply into the pharynx, pointing it at the same time more obliquely downwards. Throwing the head back raises the larynx and hyoid bone, the consequence of which is that the anterior angle of the glottis is also elevated and the mirror allowed to be still further depressed. If notwithstanding the adoption of these expedients the examiner is still unable to see the point of insertion of the vocal cords the patient should be asked to make a series of deep inspirations and rapid and complete expirations; this will raise the epiglottis against the base of the tongue and so expose the parts below, though each view lasts only for an instant.

The vocal cords are also sometimes covered by tough, clear mucus, which may prevent any proper survey of these and the neighbouring parts being made; a few slight coughs will usually expel this mucus.

Above and to the outside of the vocal cords, and running parallel with them, lie the *false* or *superior vocal cords*, distinguished from the true vocal cords by their *pale reddish colour*. Between the superior and inferior cords there is situated on each side an elongated fissure of variable breadth, the *ventricles of the larynx*, or *ventricles of Morgagni*.

It is usually a matter of considerably greater difficulty to bring clearly into view the *aryteno-epiglottidean folds*, two thin folds of mucous membrane of exactly the same colour as the rest of the mucous surface, passing from the sides of the epiglottis to the arytenoid cartilages; they are often hidden by the overhanging epiglottis. Towards their posterior ends small cartilaginous nodules are sometimes found, the *cartilages of Wrisberg;* frequently, however, these bodies are wanting.

To the outside of the cartilages of Wrisberg and Santorini are seen the *pyriform sinuses*, formed on each side by the arching outwards of the walls of the pharynx; in phonation these sinuses are dilated to some extent and are therefore then more readily made out.

When the illumination is good, and during deep inspiration, the parts below the glottis may become so distinctly visible that the cartilaginous rings of the *trachea* may be counted, particularly if the larynx be exceptionally wide. The *bifurcation* of the trachea is about the lowest point to which it is possible to see even under the most favourable local conditions; nevertheless in a few rare cases a view has been obtained of a portion of the principal bronchi.

When by repeated examination in the healthy subject a clear conception has been formed of the shape, mobility, and colour of the various parts within the larynx, one is in a position to recognise readily those deviations from the normal type which are so often met with in the diseases of the part, whether these diseases are uncomplicated and limited to the larynx or arise in connection with affections of other organs, especially of the organs of respiration. The laryngoscopic appearances presented by the most important of these affections will now be considered.

ACUTE CATARRH OF THE LARYNX.

(Acute Laryngitis).

Catarrh (or inflammation) of the larynx presents the same appearances as catarrh of the other mucous membranes: redness (vascular injection, sometimes punctate extravasations of blood), softening, and swelling. The submucous tissue is also usually to some extent involved in the inflammatory process.

The catarrh may affect, in greater or less intensity, *every* part of the larynx, or it may be *limited* to particular parts, such as the epiglottis, the arytenoid cartilages, or the vocal cords. In the latter case, therefore, we are justified in speaking of an epiglottitis, arytenoiditis, or chorditis.

Inflammation of the *epiglottis* is characterized by a deep red coloration, more frequently of its posterior than its anterior surface, and sometimes also, when the inflammation is intense, by a greater or less degree of swelling. Inflammations of the posterior wall of the larynx and of the mucous membrane which covers the *arytenoid cartilages*, are exceedingly common. The last-mentioned structures swell up very considerably, till they may be as large as peas or small beans, and become deep red in colour; their mobility in inspiration and expiration may also be diminished.—The *superior* or *false vocal cords* are also often the seat of inflammatory swelling, when they partly hide the true vocal cords; the latter then appear narrower than usual, and show only a small portion of their margin during phonation. The inflamed superior vocal cords also encroach on the ventricles of the larynx or may even obliterate them.—But of all the parts within the larynx the true *vocal cords* exhibit the most striking changes when inflamed; normally white, with a bright tendinous lustre, they become more or less deeply injected, and vary in colour from light rose to dark red, which may be spread uniformly or irregularly over their surface. Both cords are generally affected at once, inflammation of only one at a time being comparatively rare. Inflammation of moderate intensity is marked merely by vascular injection; in the more severe forms *swelling* is also observed. The inner margins of the cords are then more rounded, less sharply defined than normally, and their mobility is impaired, from loss of power in the inflamed intra-laryngeal muscles.

Inflammation of the parts within the larynx is occasionally attended by desquamation of the superficial layers of the epithelium and the formation of superficial catarrhal ulcers, particularly on the edge of the epiglottis, on the aryteno-epiglottidean folds, and on the vocal cords. The inflamed parts are generally coated with mucous secretions, which are often drawn out into fine viscous threads, especially between the vocal cords during their respiratory movements.

CHRONIC CATARRH OF THE LARYNX.

(Chronic Laryngitis).

This affection may be primary, that is, it may be developed out of an acute catarrh, or may be simply chronic in its course from the outset; or it may be secondary, appearing as a complication in destructive diseases of the larynx; or it may be of the nature of a local manifestation of some other disorder, such as syphilis or pulmonary phthisis. It is distinguished laryngoscopically from the acute form of the affection by the less bright, dirty greyish-red colour of the inflamed parts; as in acute catarrh, however, the intensity of this coloration varies from light to dark red. Like acute laryngitis, also, chronic laryngitis sometimes involves uniformly the whole of the laryngeal mucous membrane, while at other times it is confined to certain parts, such as the epiglottis, the arytenoid cartilages, the superior and inferior vocal cords, or occasionally even to one of the true vocal cords. As marked differences are observed in the degree and distribution of the swelling as in the intensity of the inflammatory redness.—Round, superficial ulcers are more common in chronic than in acute catarrh.

CROUP OF THE LARYNX.

Laryngoscopic examination is seldom called for in laryngeal croup, the symptoms of the affection being so characteristic and such as to define it so clearly from other diseases of the larynx. Apart from the painfulness of the proceeding, it is scarcely likely that any attempt to explore the larynx in this way would be successful, as croup usually occurs in children of 2—7 years of age. In such cases as have been examined the false membranes were seen, accompanied by thickening and diminished mobility of the vocal cords and consequent stenosis of the larynx.

DIPHTHERITIS OF THE LARYNX.

Diphtheritis is rarely developed exclusively in the larynx; it generally begins in the pharynx and on the tonsils, and from these parts spreads downwards.

Very young children, who are most frequently attacked by laryngeal diphtheritis, can very seldom be subjected to laryngoscopic examination; the affection, however, can usually be diagnosed without it, as we know from experience that symptoms of stenosis of the glottis (crowing and prolonged inspiration), and hoarseness or aphonia, when they present themselves along with diphtheritis of the pharynx, are always due to an extension of the disease to the larynx. Even when the pharyngeal diphtheritis is wanting the above-mentioned indications, when observed in young children in districts in which diphtheritis is prevalent in an epidemic form, generally warrant one in assuming confidently the diphtheritic nature of the laryngeal affection.

PHTHISIS OF THE LARYNX.

Pulmonary phthisis is very often accompanied by more or less extensive laryngeal disease, which may take any of the many forms to which the larynx is subject, from simple catarrh to ulceration and destruction of tissue.

Phthisical laryngeal catarrh is in no respect different in appearance from ordinary primary catarrh.—*Inflammation of the submucous tissue* adds considerably to the gravity of the disease; when this takes place the mucous membrane becomes not only hyperæmic but thickened, thrown into folds, covered with irregular rounded prominences (like those so often seen in the pharynx), and occasionally markedly œdematous.—As the phthisical disease advances the surface of the larynx is very frequently broken by *ulceration*. Ulcers of this kind may be solitary or multiple; when crowded close together they may coalesce and form one large ulcer. While they may occur in any part of the larynx they are more often met with on its posterior wall and on the epiglottis,—most often of all, however, on the *vocal cords*, usually both. The ulcerative process may attack only the superficial tissues or extend also to the deeper layers. The cords, when ulcerated, are generally reddened and swollen, and seldom of their natural colour and structure. If the loss of tissue be considerable and the ulcers be situated on the inner border of the vocal cords, the latter have a notched appearance, and when, during phonation, they are drawn together, slight irregularities are observed in the outline of the rima glottidis. In extreme cases one or other of the vocal cords may be almost totally destroyed. Laryngoscopically the loss of substance occasionally seems greater than it really is: thus, all that is sometimes seen of the vocal cords is a narrow ulcerated border; but this diminution in size is to a great extent merely apparent and caused by the undue prominence of the swollen superior vocal cords, which then conceal the true cords situated beneath them. If both vocal cords be deeply hollowed out by ulceration, closure of the glottis may be very incomplete during phonation; a gap of greater or less size will remain between the cords.—Phthisical ulcers of the vocal cords have exactly the same *shape* as ulcers due to any other cause. They are irregular and indented at the edges; if very superficial and perhaps also covered by clear tough mucus they are very difficult of detection with the laryngoscope, but when the mucus is removed, as by coughing, their dull greyish-white colour enables the observer to recognise them at once. Ulcers of greater depth or which cover a larger surface, are seen very readily and are often coated with purulent secretion.

The above-described forms of phthisical disease of the larynx are in general scarcely distinguishable, by their laryngoscopic characters, from the same forms of primary laryngeal disease. Very frequently the phthisical nature of the laryngeal affection is doubtful till the diagnosis is confirmed by the discovery, on examining the chest, of phthisis of one or both lungs. But the larynx may be the seat of true phthisical disease at a period when the physical signs of such an affection are still wanting in the lungs. In these circumstances the following points may be held to indicate, with considerable constancy, the phthisical or non-phthisical origin of the laryngeal disease; simple catarrh of the larynx disappears spontaneously, as we learn from daily experience, in a very short time, on the adoption merely of a few simple precautions; phthisical catarrh, on the other hand, is very rebellious to treatment and shows a great tendency to relapse. Phthisical ulceration in particular obstinately resists all therapeutical measures and tends rather to spread and to deepen, while catarrhal, non-phthisical ulcers usually heal very rapidly.

The order in which the various affections of the larynx appear in phthisis bears no fixed relation to the progress of the disease in the lungs; in many cases they are absolutely wanting throughout the whole course of the pulmonary phthisical disorder. On the one hand, a very advanced lung affection may be associated with simple catarrh or slight ulceration of the larynx; or on the other, the ulcerative process may rapidly extend, there may be considerable loss of substance of the vocal cords, denudation of the arytenoid cartilages, &c., while the pulmonary disorder is still in a comparatively early stage.

Syphilis of the Larynx.

As in phthisis, so in syphilis, every variety of laryngeal affection is observed.

Syphilitic catarrh of the larynx and *syphilitic inflammation of the submucous tissue,* occur generally in the same parts and present precisely the same appearances as the simple, primary or phthisical inflammations. No specific character therefore can be assigned to these affections till other signs of syphilis are discovered (in the fauces, skin, &c.).

Syphilitic laryngeal disorders, when they have lasted some time, are invariably complicated by the formation of *ulcers.* These lesions are found most commonly on the vocal cords, next most frequently on the epiglottis, especially on its free borders, and more rarely on the posterior wall of the larynx. They are distinguished from catarrhal ulcers, which are generally formed very rapidly, by their slow rate of development; laryngoscopically, however, they often offer no sign which indicates positively their syphilitic nature. In some cases, nevertheless, syphilitic ulcers differ from those of catarrhal or phthisical origin in being more nearly circular in form, in the fatty or lardaceous appearance of their floor, and in having projecting and sharply-defined edges.

The healing of the ulcers under antisyphilitic treatment is naturally the surest proof of their syphilitic character. They heal by cicatrization; where therefore such cicatrices are observed at various points within the larynx it may be assumed with almost perfect certainty that the ulcers which preceded them were syphilitic, as phthisical ulcers, which are practically the only lesions from which they need to be differentiated, as a general rule show no inclination to heal. *Cicatrices* of any great magnitude in the neighbourhood of the *glottis* give rise to deformities and impairment of the mobility of the vocal cords, and to a form of *stenosis of the rima glottidis,* which in certain cases (I have seen one such) may become so extreme as to demand the performance of tracheotomy. If from neglect, or because their syphilitic nature was not recognised, the ulcers do not heal, they may in their further progress produce the most widespread destruction of the various parts of the larynx, denudation and erosion of the cartilages (the epiglottis, in particular, being sometimes reduced to an irregular, shapeless mass), and destruction of the vocal cords so extensive that only a few almost unrecognisable remnants of them may be left.

It has been placed beyond doubt, by some careful observations, that syphilitic gummata are occasionally, though rarely, developed in the larynx. They may occur in any part of the larynx, especially in those parts situated above the glottis, in the form of circumscribed tumours, sometimes solitary, at other times present in numbers, and varying in magnitude from the size of a pin's head to that of a pea; or the gummatous matter may be diffused in the tissues, and so give rise to irregular nodulation or wrinkling of the surface. When solitary they are rounded in shape, and in aspect are sometimes yellowish, or of exactly the same colour as the rest of the mucous membrane. The difference in colour depends on the stage which the gumma has reached: in the first stage, that of infiltration (inflammatory swelling), the part is red; in the second stage, that of softening, the yellow colour begins to show itself. If the morbid material be absorbed a depression or a cicatrix is formed, or if it break up and become disorganized ulceration takes place (Scheele, &c.).

PERICHONDRITIS OF THE LARYNX.

Perichondritis, with the subsequent accumulation of purulent exudation beneath the perichondrium, the eventual escape of the pus and denudation and necrosis of the cartilage, may attack any of the cartilaginous structures which form part of the larynx. Perichondritis of the larynx is generally a secondary, rarely a primary affection, and very often follows phthisical and syphilitic laryngitis, the ulcerative process so begun extending to the deeper tissues; not unfrequently it appears in the course of severe acute diseases, particularly typhus, sometimes also in diphtheritis of the larynx and small-pox.

Until the pus collected between the cartilage and perichondrium has forced its way through the latter structure the signs discoverable by the laryngoscope do not enable one to make the diagnosis with any degree of confidence. At those parts at which the abscess is forming there is observed a firm, dark red prominence, surrounded by inflammatory swelling and projecting into the cavity of the larynx, often producing in this way a considerable degree of laryngeal stenosis; but it must be remembered that a moderately acute inflammation of the submucous tissue, particularly if developed with great rapidity, may present precisely the same laryngoscopic appearances.

Perichondritis often attacks the posterior portion of the *cricoid cartilage*, from which it rapidly extends to the other cartilages, especially in typhoid fever; if suppuration supervene and an abscess be rapidly developed, laryngeal stenosis and the phenomena of threatening suffocation are produced. The occurrence of perichondritis cricoidea in typhoid fever is usually readily diagnosed on taking into consideration the whole course of the disease in the case in hand; laryngoscopically, also, it is generally easily recognised by the firm prominent tumour which projects downwards from the posterior wall of the larynx.

Perichondritis of the *thyroid cartilage* seldom occurs primarily (except after injuries), but as a rule secondarily, in connection with the morbid conditions mentioned above. On the whole it is rarer than perichondritis of the cricoid cartilage. It may be unilateral or bilateral, strictly circumscribed or spread over a comparatively large area. If the inflammation have its seat on the outer surface of the cartilage the diagnosis is easy; most commonly however it takes place on the inner surface, being often limited exactly to the thyroid notch, when phthisical ulceration at the anterior angle of junction of the vocal cords extends to this part. Perichondritis confined to the thyroid notch is not recognisable by laryngoscopy. As it is also usually preceded or accompanied by other diseases of the larynx (inflammatory swellings, ulceration) the laryngoscopic appearances vary with the intensity and distribution of these morbid processes.

Perichondritis of the *arytenoid cartilages*, usually of both, is the most common form of the disease, and is often observed alone, unassociated with any affection of the other cartilages. In most cases it is of syphilitic or phthisical origin; in phthisis, particularly, when the laryngeal disorder has lasted some time, the ulceration frequently extends to the arytenoid cartilages and partially or totally destroys them.—In perichondritis of one of the cartilages, before the pus formed has escaped and whilst nothing is yet to be seen but swelling of the cartilage, the appearance of the parts is in all respects the same as that presented in cases of swelling of their submucous tissue, such as is so often met with in simple laryngitis. In the affection under consideration, however, the mobility of the cartilages and of the vocal cords of the same side is somewhat diminished, which is the more striking as the cartilage of the opposite side is healthy and freely movable, whilst in simple swelling of the mucous covering of the arytenoid cartilage the movements of the part are but very slightly restricted.—When the pus escapes the cartilage lies exposed; if the denudation be merely partial and circumscribed it may be completely concealed from view by the swelling of the surrounding soft parts; but if no such swelling is present, and the cartilage be separated from its connections by the destructive process, there is produced a deep depression which occupies the place of the destroyed cartilage, a change which is particularly noticeable when the corresponding vocal cord is intact.

The various forms of ulceration or necrosis of the cartilages of the larynx,—simple, syphilitic or phthisical,—are indistinguishable from each other by their laryngoscopic characters alone; the differential diagnosis rests solely on the ascertained nature of the coexisting general affection.

ŒDEMA OF THE LARYNX.

Œdema of the larynx may be either partial and circumscribed or may involve many of the laryngeal structures. It is most marked at those parts at which the submucous tissue is most lax and most capable therefore of accommodating a large amount of dropsical effusion: these parts are the aryteno-epiglottidean ligaments, the arytenoid cartilages, the posterior surface of the larynx, and the epiglottis. The œdematous swellings present a light yellow or pale reddish coloration, which is strikingly different from the deep redness of the tumefaction due to simple inflammation.

The severer forms of œdema give rise to such intense dyspnœa that careful laryngoscopic examination becomes impossible, and the swelling of the epiglottis also, which is seldom wanting in these cases, is usually so great that the intralaryngeal structures are completely hidden from sight. If the epiglottis be highly œdematous its swollen condition may usually be observed without the aid of the laryngeal mirror, by simply depressing the tongue, as in such circumstances it rises towards the base of the tongue; the tumefaction is also always easily detected by introducing the finger and passing it over the parts. A high degree of œdema of the intralaryngeal structures, particularly of the superior or inferior vocal cords, or of the aryteno-epiglottidean ligaments, always produces considerable stenosis of the rima glottidis. Less marked œdema, which for obvious reasons is more frequently met with, lessens the mobility of the arytenoid cartilages and vocal cords, particularly when the effusion is accumulated chiefly in the submucous tissue of these and the neighbouring parts. This slight partial œdematous infiltration is very often seen accompanying inflammatory ulceration.

Œdema of the larynx is invariably *secondary:* it appears as a complication of the most diverse forms of disease of the larynx (particularly perichondritis) and sometimes also of the pharynx. Sometimes, though on the whole rarely, it is connected with general nephritic and scarlatinal dropsy.—As dropsical effusions within the larynx, when they attain a certain intensity, always extend ultimately to the glottis, they are generally designated by the collective term *œdema of the glottis*.

Morbid Growths in the Larynx.

These are most commonly (in at least 75 per cent. of the cases) seated on the vocal cords, and are comparatively seldom seen at other parts of the larynx.

They may be arranged in two groups, according to their histological structure: the first includes those composed of elements which occur *normally* in the larynx, benign growths; the second those consisting of *heterogeneous* tissue elements, malignant growths.

To the *first* group belong the papillomata, fibromata, myomata, and cystic tumours of the larynx; to the *second* the different varieties of carcinoma.

The most common of the new formations developed within the larynx are the fibroid and papillary growths.

Fibroid laryngeal growths are of moderately firm consistence and, though occasionally sessile, are generally connected with the part from which they spring by a distinct pedicle. They are rounded or oval in shape, vary in bulk from the size of a pea to that of a hazel-nut, have usually a smooth, but sometimes a granulated or even lobulated surface, and are whitish, pale yellowish-red, or vivid red in colour. The differences in colour depend on the greater or less vascularity of the tumour. Fibrous growths ordinarily spring from the upper surface of the vocal cords and from their inner borders, and in the latter case may be found attached to any part of the margin; not unfrequently they are seated at the anterior insertion of the cords, and when of small size the observer has considerable difficulty in seeing them in this situation. They are seldom met with at other parts of the larynx. Fibrous tumours of the larynx are developed from the submucous connective tissue.

The *papillomatous* growths are formed by hyperplastic development of certain portions of the mucous membrane with simultaneous hypertrophy of their epithelial covering. They assume the most diverse forms: they may be elongated or rounded, pedunculated or flat and sessile, of a warty or grape-like appearance, or may occasionally resemble closely a strawberry or raspberry. Their colour is a dull white, or whitish-yellow when they contain a large proportion of fatty tissue, or pale reddish when they are more richly supplied with blood-vessels.

The surrounding parts may be healthy, or may present the signs of active inflammation. Papillary tumours most commonly grow from the upper surface and inner borders of the vocal cords; they may be situated, however, on the under surface, when it is somewhat difficult to form any estimate of their actual size, even when they project beyond the margin of the cords. They are seldom observed on the superior vocal cords, the ventricles of the larynx, or other parts. In phthisical laryngitis a number of small greyish-white excrescences of papillary aspect are sometimes seen on the posterior wall of the larynx between the arytenoid cartilages, scattered over the swollen, inflamed, and possibly also ulcerated mucous membrane, but these are not regarded as new formations in the strict sense of the term.

Cystic tumours of the larynx are rare; they are of small size, and occur most usually on the superior vocal cords and the ventricles of the larynx, occasionally also on the inferior vocal cords and the epiglottis.—*Lipoma* and *myoma* have been observed in the larynx in only a few exceptional cases.

CARCINOMATOUS GROWTHS IN THE LARYNX.

Cancerous growths are very rarely developed primarily in the larynx, or unassociated with malignant disease elsewhere; they generally extend to the larynx from neighbouring parts, the pharynx, œsophagus, &c. As the tissues so invaded are generally so altered in aspect as to be unrecognisable, and as the cancerous growth is usually of some considerable size and its rate of increase more rapid than that of any other new formation, the laryngoscopic appearances of the parts attacked scarcely admit of description. Growths of this kind have a very rough, cauliflower-like surface, which feature alone, especially if the growth be extensive, is sufficient to indicate clearly their carcinomatous nature; the diagnosis becomes more certain when similar new formations are discovered in the adjoining parts, such as the root of the tongue, or when the lymphatic glands are found to be affected by cancerous degeneration and the other signs of malignant disease are observed in the patient. When such tumours are large they naturally cause a certain degree of stenosis of the larynx.

The forms of malignant growth described as occurring in the larynx are *epithelial* and *medullary cancer*. The first is the more common variety, and that also which appears primarily and sometimes remains localised for some considerable time in this situation.[*] When epithelial or medullary cancer has spread from the pharynx to the larynx the epiglottis is generally the first part attacked by the disease.

Foreign bodies in the larynx, when not, as usual, expelled by coughing, excite most intense dyspnœa and a distressing feeling of suffocation. They may be caught and retained in the folds of the mucous membrane or may be impacted at any part within the larynx, such as the ventricles of Morgagni. The largest objects that have been removed from the larynx are sets of artificial teeth; several instances of this kind are on record.

SPASM AND PARALYSIS OF THE VOCAL CORDS.

Temporary closure of the glottis takes place from spasm of those intrinsic laryngeal muscles which approximate the vocal cords (spasmus glottidis); it occurs most frequently in children, and next most frequently in the hysterical attacks of adults. The closure may be complete or incomplete.

PARALYSIS OF THE VOCAL CORDS.

Paralysis of the vocal cords is due to a great variety of causes. Two forms of the affection are recognised, *central* and *peripheral* paralysis: the first of these is very rare, being observed in connection with cerebral apoplexy † at the part at which the vagus nerve has its origin, while the second is exceedingly common.

Peripheral paralysis of the vocal cords may be *neuropathic* (that is, caused by interference with the motor nerves of the larynx such as to deprive them of their conducting power), or *myopathic*.

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[*] I have observed one case of epithelial cancer of the epiglottis, in which the free border of the latter was transformed into an irregular, shapeless mass, which was of sufficient size to produce considerable stenosis of the larynx; in the interior of the larynx nothing abnormal could be seen.

† I have had under observation a case of apoplexy in which complete paralysis, of central origin, of the *left* vocal cord, together with paralysis of the *right* hypoglossal nerve and slight facial paresis, were suddenly developed, without any other sign of disturbance of motor power.

The *neuropathic* paralyses are generally of mechanical origin, being commonly produced by pressure on the inferior laryngeal nerve or the vagus by large tumours of the thyroid gland or of the cervical or bronchial glands, by tumours of the mediastinum and aneurisms of the aorta; in the last-mentioned case the paralysis always affects the *left* recurrent laryngeal nerve.—Neuropathic paralyses of the vocal cords occur also in certain infectious and zymotic diseases (diphtheritis, typhus, &c.) and in hysteria.

In the great majority of instances, however, paralysis of the vocal cords is *myopathic*. Two varieties have been distinguished from each other: 1. paralysis of the vocal cords in the proper sense of the term, that is, the form in which the immobility of the cords depends solely on paralysis of the muscles, the laryngeal structures being in other respects normal; 2. paralysis due simply to mechanical conditions, in which the loss of motility is caused by other laryngeal diseases, such as a high degree of swelling of the vocal cords and the parts in immediate relation with them, cicatrization after ulceration, &c. Cases of the latter kind are characterized, not by complete paralysis, but rather by greater or less impairment of the mobility of one or both cords; they have already been under consideration on a preceding page.

The paralysis may involve one cord or both; it is sometimes incomplete (paresis), at other times complete (paralysis).

In order to the better understanding of the laryngoscopic appearances in the various forms of paralysis of the vocal cords the actions of the different intrinsic muscles of the larynx may here be shortly stated.

There are three groups of muscles which move the vocal cords:

1. The muscles which separate the arytenoid cartilages (and with them the vocal cords) from each other, and which in this way *dilate* the glottis, as in inspiration; the *posterior crico-arytenoid muscles* have this action.

2. The muscles which approximate the arytenoid cartilages (and with them the vocal cords) to each other, and so *contract* the glottis. The principal muscle of this group is the *transverse arytenoid muscle*, whose action is aided by that of the *oblique arytenoid muscles*. The glottis is also contracted by the *lateral crico-arytenoid muscles*.

3. Muscles which *stretch* and render tense the vocal cords, and so tend also to bring them closer to each other. To this group belong the *crico-thyroid* and *thyro-arytenoid muscles*.

UNILATERAL PARALYSIS OF THE VOCAL CORDS.

The *left* vocal cord is much more frequently paralysed than the right. If the loss of power be complete, all the muscles which act on the cord, the dilators, constrictors, and tensors of the glottis, being paralysed together, the affected cord and its corresponding arytenoid cartilage remain perfectly immovable during respiration and phonation; the unaffected cord, on the contrary, particularly when the higher notes are sounded, moves unusually actively, and crosses over the middle line even as far as the paralysed cord, and in this way usually completely closes the glottis. The movements of the sound cord may indeed be so energetic that the corresponding arytenoid cartilage not merely touches that of the opposite side but passes over it some little distance. In all cases in which the unaffected cord comes into contact with its fellow the rima glottidis is directed obliquely towards the paralysed side. On sounding a deep note, which does not require for its production such a degree of tension of the healthy vocal cord, the latter does not cross the median line so far as to reach the paralysed cord; the closure is therefore then imperfect, a small gap being left between the vocal cords.—When the paralysis has lasted some time the cord loses its shining tendinous appearance, becomes dull and lustreless and somewhat wrinkled on the surface, and the arytenoid cartilage to which it is attached grows paler in colour and atrophies slightly.

The above-described more or less complete paralysis of all the muscles of one of the vocal cords is the most common form of unilateral paralysis and is very easily recognisable.—Paralysis restricted to certain muscles is more seldom met with. If it be confined to the dilators of the glottis the vocal cord of the affected side remains fixed in inspiration; phonation, on the other hand, is not interfered with, as the muscles which contract the glottis and stretch the cords are intact; closure of the glottis is thus normal and complete and both cords vibrate freely. When the paralysis is limited to the tensors of one of the vocal cords the movements of the cord in respiration and phonation are natural and normal, that is, the closure of the glottis is perfect; the affected cord, however, does not vibrate in phonation, it tends to curve outwards and shows on its border a *boat-shaped depression*, from deficiency of tension longitudinally (Navratil). If the paresis of the tensors of the vocal cords be bilateral the glottis becomes *elliptical* in phonation. Unilateral paralysis of the tensors of the glottis, when it appears as part of a complete paralysis of one of the vocal cords, may also be recognised by the *absence* or comparative feebleness of the sensation of vibration which should be felt on placing the finger on the outside of the thyroid cartilage, on the side corresponding to the affected cord (Gerhardt).

Paralysis of the *epiglottis* is an exceedingly rare affection (Leube, Gerhardt); if it be unilateral, produced by paralysis of either of the superior laryngeal nerves, the epiglottis is stated to be turned obliquely towards the sound side, while the upper part of the interior of the larynx, on one side, is devoid of sensation.

Bilateral Paralysis of the Vocal Cords.

Bilateral paralysis of the muscles which act on the vocal cords, unlike unilateral paralysis, is usually complete. There are two forms of the affection:

1. Bilateral paralysis of the constrictors and tensors of the glottis; and 2. Bilateral paralysis of the dilators of the glottis.

If the whole group of constrictor muscles on both sides be paralysed the vocal cords are almost motionless during inspiration and expiration, and in phonation move only very slightly towards the median line; even in forced phonation they never come perfectly into contact with each other, or if they do for an instant touch they at once fall asunder again. The vibration of the cords is also very imperfect, as the muscles which draw them together, the constrictors of the glottis, are also to some extent tensors of the vocal cords. If the paralysis of the constrictors be incomplete the movements of the vocal cords in respiration and phonation are not entirely abolished, though they are much less extensive than normally; a great many intermediate stages between absolute immobility and normal freedom of movement are observed.—Bilateral paralysis of the constrictors of the glottis may also be partial in this respect, that the loss of power may be limited chiefly to the lateral crico-arytenoid and the thyro-arytenoid muscles, or to the transverse arytenoid muscle. The following varieties of this partial paresis are met with:

a. The anterior angle of junction of the vocal cords is closed and the arytenoid cartilages touch each other in phonation, while the other parts of the cords make no movement towards each other, the *middle* portion of the rima glottidis, the pars ligamentosa, remaining open; this condition indicates paralysis confined chiefly to the lateral crico-arytenoid and the thyro-arytenoid muscles.

b. The ligamentous portion of the vocal cords closes normally, but the cartilaginous portion continues open, the arytenoid cartilages remaining fixed; here the paralysis affects principally the transverse arytenoid muscle.

c. The vocal cords come together only in their anterior part, the rest of the ligamentous and the whole of the cartilaginous portion remaining open.

Bilateral paralysis of the *dilators of the glottis* is much less common than that of the constrictors. It presents the following laryngoscopic appearances: the vocal cords, even in quiet respiration, are abnormally close together, the rima glottidis being reduced to a mere chink between them. In inspiration the cords are not drawn apart; they are, on the contrary, approximated to each other by the diminished pressure of the air, the glottis is almost completely closed, extreme dyspnœa is established, and inspiration acquires a whistling, sonorous character which is quite distinctive of stenosis of the glottis. In expiration the cords are again separated slightly, but only so far as to leave the above-described narrow chink through which the air may pass. In phonation the vibration of the vocal cords, and the movements of these structures and of the arytenoid cartilages towards each other, are normal, the constrictors of the glottis and the tensors of the vocal cords being intact.

Apart, perhaps, from the appearances observed in complete unilateral paralysis of the vocal cords, there is no more striking group of signs connected with motor disorder of the parts within the larynx than that just described, associated with bilateral paralysis of the posterior crico-arytenoid muscles. Although the number of cases of this form of paralysis recorded in medical literature is up to the present time rather small, it seems not to be so rare an affection as might on that account be supposed; I have seen two such cases, both patients being hysterical.

In most cases of paralysis of the vocal cords there is more or less change in the voice, hoarseness of all degrees of severity up to complete aphonia, or simply difficulty in the *formation of sounds*, observable sometimes only in the upper notes, sometimes only in the lower. The loss of voice is complete when the vocal cords and the arytenoid cartilages are not properly approximated, a space of variable breadth being left between them even during the most strenuous efforts to produce a sound; the voice fails also, even though the closure of the glottis be complete, if the cords have lost their power to vibrate, from paralysis of the tensors of the glottis. If the glottis close perfectly, however, and if the paralysed cord vibrate normally, the strength and purity of the voice may be preserved unaltered. But if the cords do not come fully and accurately into apposition, if, for instance, the glottis be completely closed in its ligamentous but not in its cartilaginous portion, the voice becomes hoarser.—With regard to the difficulty in the formation of sounds, or alteration or failure of the sound when the voice is of a certain pitch, the most diverse modifications are observed; the only general statement that can be made on the subject is that the number of sounds that can be uttered by the patient and their purity depend—provided always that notwithstanding the unilateral paralysis of the vocal cords efficient closure of the glottis takes place—on the amount of vibration of which the affected cord is capable. Thus, the voice very readily breaks into falsetto when only the inner margin of the paralysed cord enters into vibration; it, on the other hand, becomes deeper in pitch when the cords are shortened and relaxed, from paresis of the thyro-arytenoid muscles, a form of the affection which is not uncommon. When energetic attempts at phonation are made sounds may naturally still be produced which are not heard as the result of feebler efforts, as in the former case more accurate closure of the glottis takes place and the vocal cords are thrown into more active vibration. *Diphthonia* is sometimes observed when the tension of the vocal cords is unequal on the two sides.

In all other affections of the larynx, from simple catarrh to the destructive ulcerative processes, temporary or permanent impairment of the voice, varying in intensity from time to time, amounting in some cases merely to slight hoarseness, in others to complete aphonia, is a symptom almost as constant as cough in the various diseases of the respiratory apparatus. But just as the cough cannot be said to be characteristic of the special *nature* of the pulmonary disorder, so the alteration in the voice indicates only approximately the nature, seat, severity, and extent of the laryngeal affection.

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